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THE ENZYMATIC DEBRIDEMENT OF WOUNDS*

A PRELIMINARY REPORT

A. Gibson Packard, Jr., M.D.

Baltimore, Md.

This is a preliminary report on the use of Papain,† a proteolytic enzyme for topical use as an agent for surface debridement. Papain occurs in the latex of the fruit of Carica papaya. The enzyme is supplied so that upon final dilution, a 50 cc. solution contains a total of 250,000 units[1] of the proteolytic enzyme, Papain.

Irrespective of the site of the lesion to be debrided, the following protocol was used: The patient was examined initially and the extent of devitalized tissue evaluated. If necrotic tissue could be debrided mechanically this was done. The area was then flushed with 3 per cent hydrogen peroxide and allowed to dry. A piece of gauze, the approximate size of the affected area, was applied to the wound, and this gauze soaked with the Papain solution. The amount used for each particular patient varied with the size of the area. The piece of gauze then was covered with a thin transparent water proof plastic material to prevent drying by evaporation or diffusion. This was, in turn, covered by dressings and all were held in place by adhesive tape or elastic bandage. This dressing was not disturbed, as a rule, for a period of 24 hours. At the end of this period the wound was examined once again. Any loose necrotic tissue was removed and the process repeated. The program for each patient went on daily until complete debridement and a healthily granulating wound was obtained, or until the treatment had to be discontinued from some unforeseen reason (table I).

CASE REPORTS

Case 1. A. E., was a 5 year old Negro boy. The area affected was on the anterior aspect of the right leg, just below the knee. The patient had developed a hematoma in this area

* From the Department of Surgery, University of Maryland, University Hospital, Baltimore, Md.

† The Papain used in this study was supplied by the Winthrop Laboratories, Inc., New York, N. Y.

TABLE I

Case	Lesion	Date of Injury	Treatment Started	Treatment Stopped	Evaluation
1	Ant. right leg, 2 by 1.5 cm.	11–13–55	11–16–55	11–18–55	Adequate debridement
2	Ant. right leg, 11 by 2 cm.	10– 9–55	11–16–55	11–18–55	Improvement but patient uncooperative
3	Left buttock, 5 by 5 cm.	7– 1–55	11–28–55	11–30–55	Unsatisfactory during brief trial and patient unwilling to come daily
4	Ant. right leg, 5 by 5 cm.	3– 55	11–28–55	11–30–55	Good results
5	Pilonidal sinus	10–24–55	11–28–55	11–30–55	Discontinued due to severe pruritis
6	Ant. right leg, 5 by 1 cm.	10–25–55	11–28–55	11–30–55	Good results
7	Back of neck, 8 by 4 cm.	11–22–55	11–29–55	12– 8–55	Excellent result
8	Ant. chest and abd. wall	11–20–55	12– 5–55	12–13–55	Excellent result
9	Ant. abd. wall, 6 by 3 cm.	11–24–55	12–13–55	12–22–55	Excellent result
10	Post. chest wall, back, buttocks, and thighs.	11–21–55	12–13–55	12–15–55	Excellent result
11	Dorsum of hand, 6 by 2 cm.	12– 5–55	12–14–55	12–20–55	Excellent result
12	Scalp	12– 4–55	12–19–55	12–22–55	Excellent result

2 weeks prior to treatment when he was struck by an automobile bumper. The hematoma had become infected and had drained spontaneously. At the time of examination there was a small subcutaneous cavity largely filled with necrotic tissue. The opening on the surface was 2 by 1.5 cm. The initial treatment began on Nov. 16, 1955. The patient returned the following day, and there was some improvement. The dressing was reapplied and, when the patient returned the following day, complete debridement had resulted. No further application was necessary. By Nov. 30, 1955 the wound had epithelialized completely.

Case 2. M. A., was a 27 year old Negro woman. The patient had been in an automobile accident on Oct. 9, 1955. She sustained an avulsion of skin on the anterior aspect of the right leg. She had been treated in the Surgical Clinic from the period of her injury until November 16. A large amount of necrotic tissue remained in the wound, and it was thought that enzymatic debridement should be undertaken. The wound at this time measured 11 by 2 cm. Papain was applied, and the patient returned the following day. She complained vigorously of itching of the skin about the wound and was reluctant to continue the use of the solution. However, she did do so. She was expected to return on Nov. 18, 1955 but did not. A week later when she did return, the wound was granulating beautifully, and in a period of another week was well healed.

Case 3. I. B., was a 57 year old Negro woman. The patient had had radiation therapy in 1940 (for carcinoma of the cervix). Two anterior and two posterior quadrants were treated. Fifteen years later ulcerations occurred in an area of the left buttock possibly incident to trauma in that area. When seen she was found to have an ulcer measuring 5 by 5 cm. with a dirty, necrotic base. Treatment was begun on Nov. 28, 1955. The patient was seen on 2 subsequent days, but little or no improvement was obtained. She was reluctant to be treated daily, and since the results of several days' treatment were poor, it was thought that this patient should be discontinued on therapy and treated in some other fashion.

Case 4. W. M., was a 55 year old white man. The patient had developed a stasis ulcer

on the anterior aspect of the right leg. This ulcer had resulted from a contusion which occurred 8 months prior to being seen in the clinic. Papain was begun on Nov. 28, 1955. The area at this time measured 5 by 5 cm. The patient was followed for a period of 3 days. By the end of the third day adequate debridement had been obtained, and the patient was carried thenceforth on routine external supportive treatment for stasis ulcer.

Case 5. H. R., a 17 year old Negro girl, had had incision of an infected pilonidal sinus on Oct. 24, 1955. It was elected on November 28, to treat the patient with Papain inasmuch as the area in the posterior midline was quite deep and had a great deal of necrotic tissue in its base. Treatment was started on Nov. 28, 1955. The patient returned the following day and complained vigorously of itching in the areas of application. However, she was redressed with the same material. Upon returning on the following day she refused to continue the treatment because of pruritis. The treatment was discontinued. No definite evaluation as to the efficacy of the preparation could be obtained since the period of treatment had been relatively short for the area involved.

Case 6. L. W., was a 9 year old Negro girl. On Oct. 25, 1955 the patient struck her right leg. When seen on November 28, this area was represented by an ulcer measuring 5 by 1 cm. The patient, because of her youthfulness was not necessarily cooperative. She returned each day without her dressing, but definite evidence of improvement was shown. By Nov. 30, 1955 it was decided that the patient was too uncooperative to continue therapy but that she had definite improvement in her wound inasmuch as most of the necrotic tissue had disappeared. In a subsequent period of 10 days the wound granulated in and closed without further treatment.

Case 7. P. C., was a 51 year old white man. The patient had been in the University Hospital for a period of time for treatment of empyema of the chest. He subsequently developed an enormous carbuncle on the back of his neck. He was readmitted on Nov. 22, 1955. The carbuncle was incised by multiple transverse and linear incisions. In the Surgical Clinic when first seen on November 29, the area on the back of his neck measured 8 by 4 cm. He was seen daily and Papain applied. By December 4, the wound was remarkably improved and all necrotic tissue had been removed. Healing was complete without skin grafting by Dec. 30, 1955.

FIG. 1a

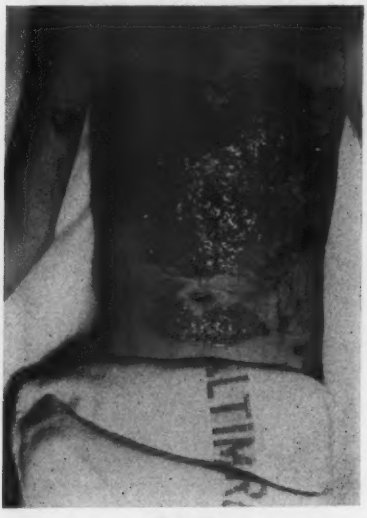

FIG. 1b

Case 8. B. R., was a 9 year old white girl who sustained second and third degree burns of the entire anterior chest wall and abdomen when her clothes caught fire (fig. 1a). Initial supportive treatment was given, and the burn covered by vaseline gauze dressings. On Dec. 6, 1955 Papain treatment was started. By Dec. 13, 1955 all burn eschar had disappeared (fig. 1b). The following day she was skin grafted.

Case 9. D. B., was a 49 year old Negro man. The patient had undergone a laparotomy on Nov. 24, 1955 for a stab wound of the abdomen. Postoperatively he developed a wound infection which resulted in necrosis of his anterior rectus sheath over an area of 6 by 3 cm. In the Out-Patient Department Papain debridement was begun on December 13. He was dressed daily for 7 days. After Dec. 22, 1955 no further debridement was necessary. At the end of the debridement period the wound measured 6 by 2 cm.

Case 10. L. Q., was a 3 year old Negro girl who was admitted to the hospital on Nov. 21, 1955 severely burned when her clothes caught on fire. The entire back from the neck to the buttocks was burned. This was largely a third degree burn which almost completely encircled the upper thighs as well. Treatment was started on December 13 (fig. 2a). By Dec. 15, 1955, it was thought that the debridement was satisfactory and that no further debridement would be necessary (fig. 2b).

Case 11. M. B., was a 70 year old Negro man who had an area of skin on the dorsum of his right hand and thumb avulsed by a circular saw on Dec. 5, 1955 (fig. 3a). The original area measured 6 by 2 cm. He was dressed daily with Papain for 7 days. On Dec. 30, 1955 the wound measured 3 by 2 cm. and was clean and granulating (fig. 3b).

Case 12. E. H., was a 4 months old Negro male infant who sustained a third degree burn of his scalp on Dec. 4, 1955. The entire vertex was involved in a third degree burn. On December 19 dressings with Papain were begun (fig. 4a). He was redressed daily until Dec. 22, 1955 when the burn eschar was sufficiently elevated so that it could be mechanically debrided. Further debridement was unnecessary since all necrotic tissue had disappeared (fig. 4b).

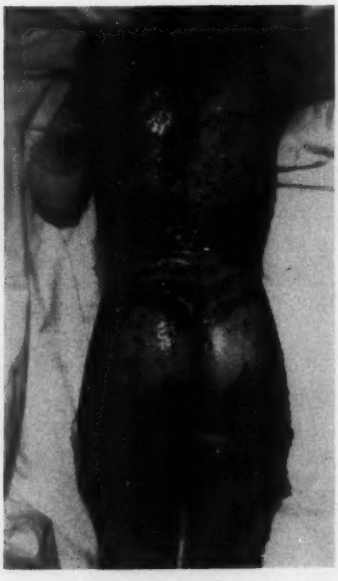

FIG. 2a

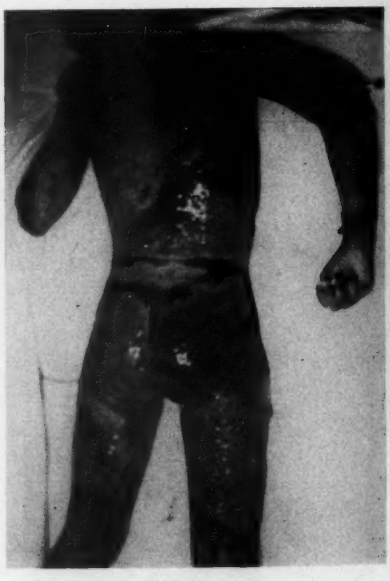

FIG. 2b

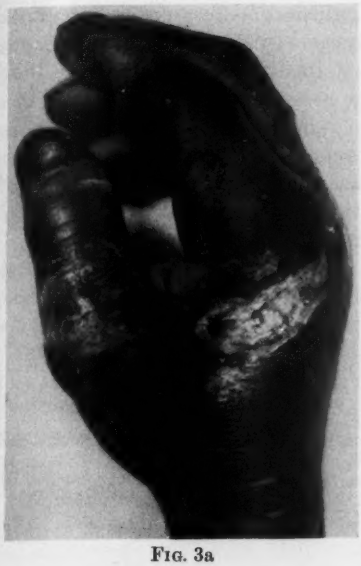

FIG. 3a

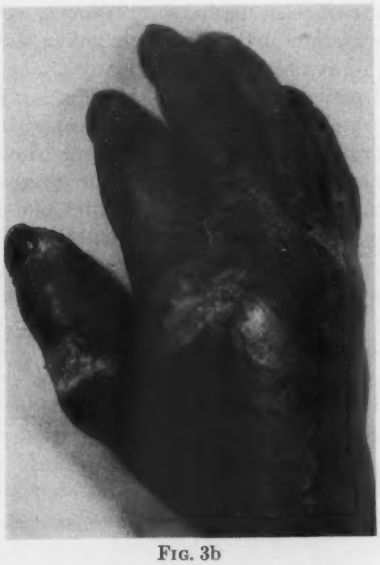

FIG. 3b

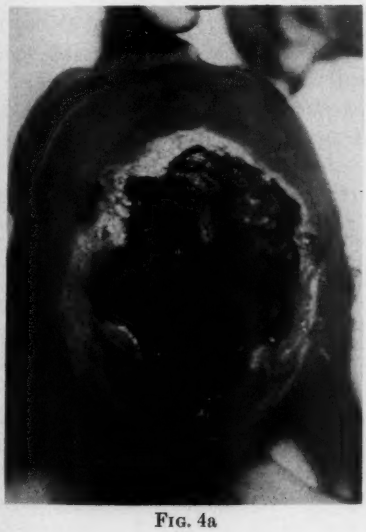

FIG. 4a

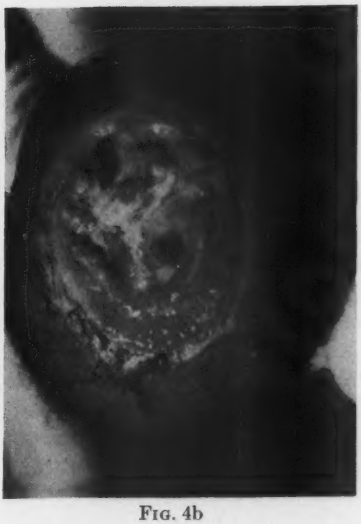

FIG. 4b

DISCUSSION

Papain appears to be an excellent and efficient agent for topical enzymatic debridement. In none of the above patients was there evidence of delay in the appearance of granulation tissue, nor was there visible evidence of irritation of

the adjacent normal skin. However, several patients noted an unpleasant pruritic sensation in the skin surrounding the area under treatment, and in 2 patients this symptom was sufficiently annoying that treatment was stopped. In these 2 patients no visible evidence of skin change was apparent. It is suggested that protection of the adjacent skin with petrolatum jelly might be sufficient for protection against this symptom when it is annoying. The use of aqueous zephiran rather than 3 per cent hydrogen peroxide as a cleansing agent is also suggested to eliminate oxidative inhibition of enzymatic activity.

Dept. of Surgery
Univ. of Maryland Hosp.
Baltimore, Md.

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ANORECTAL COMPLICATIONS OF PREGNANCY

WALTER H. GERWIG, JR., M.D.*

Washington, D. C.

Anorectal complications associated with pregnancy may be those which affect the mother and those which occur in the infant. Maternal complications are for the most part due to trauma while those seen in the newborn are the result of developmental abnormalities in which there is either failure of descent of the hindgut, failure of invagination of the proctodeum or there is formation of an ectopic opening.

Other lesions occur in the anorectal area of the mother, but in the strict sense they cannot be considered as complications resulting solely from pregnancy as they may occur in the nulliparous female or in the male. This group (hemorrhoids, fissures, cysts, etc.) even though not common to pregnancy, appear to take on somewhat different characteristics when they occur in conjunction with it. Pregnancy is not necessary for the development of any of these but it is believed that their coexistence alters the course and the management.

Unlike those conditions that result from pregnancy and/or delivery, and distinguished from those conditions which are altered by but do not necessarily result from pregnancy, we have still a third category. Under this heading, we may include such lesions as anal warts, rectal polyps, and perianal dermatitis. These do not appear to be altered in any way when they occur during pregnancy, and therefore will not be included in this discussion.

Having outlined briefly the various anorectal lesions that may result from, be altered by, or be unchanged by pregnancy, we now may proceed to consider these various conditions separately.

I. TRUE ANORECTAL COMPLICATIONS OF PREGNANCY

A. Maternal Anorectal Injuries Which Occur During Delivery: Until the past two decades it was not unusual to encounter patients who suffered from bowel incontinence, rectovaginal fistula, or a destroyed rectovaginal septum following delivery. At present such patients are seen rather infrequently, which in itself indicates the high standard of obstetrical care given to mothers today. Three factors seem to account for the lowered morbidity rate following delivery. 1. In the larger cities about 9 out of 10 deliveries take place in a hospital, where equipment, assistants and anesthesia are available. 2. The episiotomy is being performed with increasing frequency, and tends to reduce and control the possibility of laceration. 3. The immediate and apparently skillful repair of the third and fourth degree lacerations which results in almost 100 per cent satisfactory wound healing.

According to Dodek,[1] Kaltreider and Dixon,[4] and others, we may now look

* Associate Clinical Professor of Surgery, The George Washington University School of Medicine, Washington, D. C.

upon permanent anorectal damage following delivery as somewhat unusual. When severe damage has occurred and has not been corrected, it is wise to wait several months to see what degree of regression and restoration of function may occur. If none is noted, a complete fecal diverting colostomy is created and repair of the damaged sphincter accomplished. About 4 weeks later when healing has taken place and sphincter control has been re-established, the colostomy may be closed.

B. Maternal Coccygodynia: In addition to the tears into the sphincter and rectum that have been described, trauma may occur during delivery with damage to the coccyx. Coccygodynia secondary to parturition does not constitute a large per cent of cases, since there are a great many patients with coccygodynia due to falls, anorectal inflammation, etc., giving rise to the rather characteristic aching pain located in the coccygeal region which tends to radiate either into the rectum, out into the buttocks, up into the back or down the thigh. The immediate therapy is advisably one of conservatism. However, coccygectomy occasionally may be required when symptoms persist, and there is evidence of dislocation, fracture, or hypermotility of the coccyx.

C. Anorectal Complications Occurring in the Newborn: Embryologically, the rectum originates from the terminal portion of the hindgut. It is of entodermal origin, and ultimately becomes lined with columnar cell mucosa. The anus develops from the embryonic proctodeum which is of ectodermal origin and is lined with squamous cell epithelium. The normal sequence of events during fetal life is the combined descent of the hindgut associated with an invagination of the embryonic proctodeum. These structures meet at what ultimately becomes the anorectal line. At this point, a membrane is temporarily formed. This undergoes dissolution with establishment of continuity of the lower intestinal tract. Congenital lesions in this area may be due to complete absence or arrested descent of the rectum, with or without ectopic communications into neighboring structures or to the outside. The manifestation of failure of invagination of the embryonic proctodeum may be complete absence, membranous occlusion, or some degree of stenosis. It becomes relatively clear that a tremendous number of combinations of defects are possible. The diagnosis of these congenital anorectal malformations is not difficult and is made by the obstetrician immediately following delivery. Early diagnosis permits earlier correction of the condition with an ultimate improved prognosis. On examination, a membranous occlusion or imperforation of the anus may be suspected, however, sufficient delay should be accomplished to permit both the search for other congenital malformations and the passage of air down the intestinal tract. When the child is put in an inverted position and roentgenograms are taken, this forms a bubble. The visualization of this bubble permits one to rather accurately judge the degree of descent of the rectum. It is essential that both anteroposterior and lateral roentgenograms be taken, as a single view may be misleading. When the anomaly is one causing complete obstruction, an attempt at correction should be carried out within the first 36 hours. This appears to be a golden period. When there is complete absence of the rectum and failure of descent, an abdominoperineal

procedure will be necessary for correction. In the premature or poor-risk infant a complete fecal diverting colostomy may occasionally suffice as an emergency measure. In the event that descent has occurred, but is incomplete, it may be possible to do a pull-through procedure from the perineal side. When stenosis or a fistula is present, the problem of complete obstruction is not a hazard. A stenosis may be managed by dilatation, but in the great majority of patients poses no problem. Fistulization usually is best treated by fecal-diverting colostomy and an attempt to repair the fistula at a later date.

II. ANORECTAL DISORDERS NOT NECESSARILY COMMON TO PREGNANCY BUT USUALLY ALTERED WHEN PREGNANCY IS PRESENT

A. Hemorrhoids: Hemorrhoids probably can be considered the most common lesion to occur in the anorectal area. It is a lesion that is influenced by pregnancy. It has been said that they are the price that human beings must pay for having assumed an upright position. There is little doubt that mechanics play a major role in the development and progression of these varicosities. Straining with bowel movements, the development of sphincter spasm and any factors that tend to retard hemorrhoidal venous flow will aid in the production of hemorrhoids. Hemorrhoids are classified as being internal in the event they are covered with mucosa and located proximal to the intersphincteric line, or external if they are distal to the intersphincteric line, and covered by skin. Hemorrhoids show little or no concern in regards to sex, and the lack of pregnancy is certainly no safeguard against their development. It has become increasingly noticeable that when hemorrhoids are present in the female, they appear to cause more reason for complaint at the time of the menstrual period and during pregnancy. Both situations give rise to symptoms which probably will subside greatly following the cessation of menses, or following delivery in the event of pregnancy. In addition to the mechanical factors of possible pressure, stasis, etc., there appears to be some factor in the menstruating woman and in the pregnant woman which may give rise to vascular relaxation. When it is considered that the growth of the uterus during pregnancy causes elongation and enlargement of the numerous blood vessels one may speculate as to whether or not the factor which permits such elongation and enlargement may be something that either circulates in the bloodstream, or is deprived from the body at the time of menses and during pregnacy. This unknown factor, whether it be withdrawn or is created in the body, appears to have its greatest influence during pregnancy. It is difficult otherwise to understand in some pregnancies why severe hemorrhoids develop so early in the course prior to any evident enlargement sufficient to cause great pressure. Protrusion, pain and bleeding are the predominant manifestations of hemorrhoids in pregnancy. Unlike the varicosities of the lower extremity which may be controlled somewhat with the aid of elastic stockings, etc., there are no appliances that can be used. The knee-chest position which permits mechanical drainage of the hemorrhoidal area probably is the single greatest asset for the management of mild to moderate hemorrhoids during pregnancy. An exaggerated position is not necessary, the only requirement being that the anus is

elevated to a level above the heart. Local applications in the form of ointments, astringents, and suppositories may be beneficial. The management of bowel habits guided toward elimination of constipation becomes necessary.

We have recently undertaken an additional form of therapy, the results of which have been most encouraging. The method is to perform hemorrhoidectomy providing certain criteria warranting its performance be present. In our experience we have believed that the time for performance of a hemorrhoidectomy must be after the end of the sixteenth week and prior to the onset of the thirty-second week. During this interval which begins at the time of the complete fixation of the placenta, we believe that hemorrhoidectomy has shown itself to be a valuable procedure, and the outcome has been very satisfactory. We are reluctant about performing this operation after the thirty-second week because healing undoubtedly will be incomplete at the time of delivery. When the procedure is considered for the postpartum period in patients who have suffered undue stress during the late stages of pregnancy, and still possess severe hemorrhoids after delivery, we have performed the operation only after the tenth postpartum day. It is believed that this allows adequate time to see what regression might take place.

We rarely see any primiparous patients that we believe should undergo hemorrhoidectomy during the prenatal period. Most of our patients who have been operated upon have been multiparous. They have had severe pain, discomfort, and distress during the pregnancy, and their hemorrhoids did not regress. They had not had any surgical treatment for the hemorrhoids, and early in the stages of a subsequent pregnancy developed severe pain, protrusion, and/or bleeding. In such patients we then felt justified in performing a hemorrhoidectomy. By observing these people at the time of delivery we are greatly pleased with the results. All patients were operated upon under caudal anesthesia, utilizing the Sims position, which is not at all uncomfortable for them to assume. We are able to control the pain during the postoperative period by the use of a continuous caudal indwelling polyethylene catheter which permits periodic introduction of an anesthetic agent to control the discomfort.[2]

B. Fissure: The anal fissure is another manifestation not necessarily common to pregnancy but certainly influenced by the delivery. Fissures probably are seen much more frequently in the female than in the male, and their occurrence in the posterior aspect is, as a rule, much more common than their occurrence in the anterior midline. Multiple fissures do occur, but are relatively rare. It has been our experience that anterior fissures are seen for the most part following delivery. We also see posterior fissures, but the ratio of an anterior to posterior fissure following delivery is almost 1 to 1, while in the male or nulliparous female, the occurrence of fissures shows a predominance of posterior to anterior being about 9 to 1. Also of interest is the fact that in the postpartum patient the anterior fissure may well heal under conservative measures, and cause no further trouble. The posterior fissure, however, seems to give rise to the most severe pain, and the severity which is rather unrelenting more or less conditions the patient to seek early management and by so doing presents herself for surgical inter-

vention before the onset of chronic changes. It has been our custom to excise these fissures under caudal anesthesia, and at the same time do a partial sphincterotomy. Postoperative care as with most anal operations consists of control of pain, the employment of laxatives and an anticonstipating type of diet, and the use of warm tub baths or compresses. The hospitalization requires about 48 hours for the pain control method utilizing the continuous caudal anesthesia, plus an additional 48 hours to assure proper progress of healing. The patient is discharged about the fifth day and is followed thereafter as an outpatient.

C. Presacral Cystic Tumors: The final group of lesions which, again, is not necessarily common to pregnancy, but in our experience has tended to show some association, is the presacral cyst. Of 5 cases reported recently, these presacral tumors were found to be multilocular and cystic, and occurred in females. Three of the 5 histories indicated that there was an increase in size of the tumor following delivery. In 2 of the patients, the obstetrician had not discovered the presacral tumor during prenatal examination, but had picked it up following delivery. The third patient was known to have a presacral cystic tumor which increased decidedly following delivery. These are relatively uncommon lesions but it behooves the obstetrician to palpate the sacral curvature and presacral area when doing a final postpartum checkup, as he may be the first to make a diagnosis of a nonsymptomatic neoplasm in this region.

SUMMARY

Anorectal complications associated with pregnancy may be those which affect the mother and those which occur in the infant. These lesions have been considered as those resulting directly from pregnancy, and those that may occur during and complicate pregnancy, but not necessarily directly resulting from pregnancy. The management of these complications is discussed.

George Washington University
School of Medicine
Washington 7, D. C.

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ANTERIOR SACRAL MENINGOCELE

RICHARD S. SILVIS, CAPT., MC, USN,* LINDSAY R. RIDDLE, CAPT., MC, USN,†
GALE G. CLARK, CDR., MC, USN‡

Oakland, Calif.

The occurrence of an anterior sacral meningocele is so infrequent that it is rarely, if ever, considered in the differential diagnosis of pelvic masses that may obstruct the rectosigmoid colon. Aspiration, or surgical attack without diagnosis has resulted in many catastrophies in the past, because of ensuing meningitis. A review of the pertinent literature will be presented, and a concept that elective diversion sigmoid colostomy prior to the definitive operation via a sacral exposure, propounded.

The exact etiology of anterior meningoceles is conjectural, but it is agreed that their development is related to a congenital defect in the anterior sacral wall. The thinness of the latter, as compared with the bulky sacral bodies at a higher level, explains the fact that anterior meningoceles do not occur except through the sacrum. The smooth outline of the osseous defect would suggest that the original defect might have been comparatively small, and that it was symmetrically enlarged by the even pressure of the presenting meningocele.

Eder[3] reviewed the literature and tabulated 45 cases in 1949. Since then numerous reports have appeared.[1, 4, 5] The increased recognition of this rare lesion and of many others, such as annular pancreas, should reward those dedicated teachers of surgery who, through persistent endeavor, have brought surgical practice in America to its present level. A total of only 45 cases of anterior sacral meningocele had been reported by 1949, and 12 during the past 6 years. This is consistent with the progress of our teaching. Table I is a summary of cases reported. Our case is the fifty-seventh, and the eighth in a male.

It is of interest that the vast majority of the reported cases have been in adults, in contradistinction to the infant and childhood incidence of spina bifida and posterior meningocele. One might hypothesize that the lower incidence of inclusion of elements of the central nervous system in anterior meningoceles, rendering them less symptomatic than the posterior, may account for this discrepancy; or that their occult position, deep in the pelvis, may contribute to their nondiscovery.

The sex incidence (47 females/8 males) would appear to clarify the preponderance of anterior meningoceles in adults as opposed to the almost equal sex incidence of posterior meningoceles in infants. It is obvious that a congenital defect cannot occur more frequently in adults. The lesion is detected more frequently

* Chief, Surgical Service; † Surgical Service; and ‡ Head, Neurosurgery Branch—U. S. Naval Hospital, Oakland, Calif.

The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

in females, due to pelvic examinations and to the catastrophies that sometimes occur during childbirth. The following is therefore offered for consideration:

1. Anterior meningoceles, while less common than the posterior variety, are much more prevalent than supposed, and are not diagnosed (45 until 1949; 12 since then).

2. Until proved otherwise, we believe that this congenital deformity may have no uneven sex ratio, and that innumerable cases in the male go undetected.

ERRORS IN THE PAST

Women have died during childbirth as a result of sudden rupture of anterior sacral meningoceles. More regrettable are the deaths resulting from meningitis following aspiration through the rectum or vagina of what was assumed to be perirectal or pelvic abscess. A rupture of the meningocele during rectal examination has been reported.

DIAGNOSIS

The *sine qua non* is the roentgenologic finding of a scimitar sacrum; but since one should diagnose primarily on clinical findings, these will be discussed. Because the anterior bulging does not encompass nerve components, anyone can imagine the symptoms. They are essentially those of a smooth, space-filling, noninflammatory mass in the pelvis. Urinary symptoms, dysmenorrhea, dystocia, and obstipation may result. Rarely are the rami of the sacral nerves involved, resulting in paresthesia of the third, fourth and fifth toes, the lateral aspect of the lower leg, the posterior aspect of the thigh, or even of the buttock and anal fold. If the communication between the meningocele and the subarachnoid space is sufficiently large, *headache*, that is related to sitting, straining at stool, and relieved by lying down, may be the presenting complaint.

The diagnosis is established by the abdominal and rectal palpation of a presacral pelvic mass of the characteristics as outlined above, and the roentgenologic finding of a scimitar sacrum. The demonstration of a communication between the subarachnoid space and the presacral mass is conclusive evidence, but is unnecessary and probably unwise as a routine measure.

CASE REPORT

D. C. M., a 32 year old white man, was admitted to the hospital on May 4, 1954, complaining of obstipation, anorexia, nausea and cramping abdominal pain of 1 week's duration. While he had had no stool for this period, he had passed flatus. No melena or vomiting had occurred. He had had no abnormal urinary symptoms.

The past history revealed that he had suffered from chronic constipation since childhood, to such a degree that regular enemas and laxatives had been required. He had never undergone a previous episode of complete obstipation for 7 days. He had experienced frequent generalized headaches of 1 to 3 hours' duration all his life. Occasionally he had had great difficulty in urination and could accomplish this only in the sitting position. An older brother had been successfully operated upon at the age of 47 for a presacral, dermoid tumor. Eleven other siblings were in good health. Two sisters had successfully completed pregnancies.

TABLE I

Summary of reported cases of anterior sacral meningocele

Case No.	Author (Date)	Sex	Age (Yrs.)	Symptoms	Treatment	Result	Remarks
1	Bryant (1837)	F	25	Difficult pregnancy; abnormal labor	None	Accidental death	Sacral defect demonstrated at postmortem examination
2	Emmet (1871)	F	36	Constipation; dysuria	Aspiration per rectum	Died; uremia	Sacral defect demonstrated at autopsy; cauda in sac
3	Hofmokle (1878)	M	11	Mass, iliac fossa	Unknown	Died; cause (?)	Sacral defect
4	Hugenberger (1879)	F	25	Dystocia	None	Died; peritonitis	Sacral defect; cauda in sac; sac out of foramen
5	Kroner-Marchand (1881)	F	20	Constipation; abdominal mass	1. Aspiration per vagina 2. Cyst opened (?)	Died; meningitis	Bicornuate uterus
6	Thomas (1885)	F	28	Pelvic mass and discomfort	None	Unknown	Retrorectal mass; questionable case
7	Thomas (1885)	F	19	Pelvic mass; backache and dysmenorrhea	1. Aspiration, twice per vagina 2. Cyst sutured to vagina	Died; meningitis	
8	Lohlein (1895)	F	28	Constipation; pelvic cyst complicating pregnancy	Aspiration, 3 times per vagina and rectum	Cured; local infection	
9	Borst (1898)	?	3 mos.	Pelvic cysts	Excision, cysts	Died; etiology (?)	
10	Marwedel (1901)	F	13 dys.	Pelvic cysts	Excision, cysts	Cured	
11	Pupovac (1903)	F	25	Pelvic cysts	Resection per perineum	Cured	Bicornuate uterus; teratoma, anus
12	Robinson (1903)	F	11 mos.	Abdominal mass	Laparotomy; ligation, cyst	Died; meningitis	Large defect extending from T12 to L5

13	Willard (1904)	F	2 mos.	Lumbar and abdominal tumors	Unknown	Unknown	Spina bifida with anterior opening
14	Nieberding (1904)	F	18	Abdominal pain and tumor	1. Exploratory laparotomy 2. Aspiration per vagina	Unknown	
15	Neugebauer (1905)	F	20	Constipation	None—spontaneous rupture	Died; meningitis	Double uterus and vagina
16	Grossman (1906)	M	6 mos.	Constipation; dysuria; tumor, right gluteal region	Excision; ligation, pedicle	Cured	X-ray evidence of sacral defect; nerves in sac wall
17	Ehrlick (1909)	F	4	Abdominal tumor; club foot	Laparotomy; excision, cyst	Died; pneumonia	Many meningoceles through lumbar intervertebral spaces
18	Fairbairn (1911)	F	18	Abdominal pain; dysuria and pelvic mass	Excision, cyst, through perineovaginal approach	Died; meningitis	Bicornuate uterus; dilated ureters
19	Tulp (1912)	F	38	Constipation; pelvic tumor	Aspiration	Died; meningitis	Postmortem x-ray showed deformity of sacrum
20	Sawicki (1914)	F	25	Constipation; pelvic tumor	Laparotomy; cyst marsupalized	Cured	Meningocele with dermoid
21	Mayo (1915)	F	22 mos.	Pelvic tumor	Laparotomy with drainage	Died, age 22; teratoma	Teratoma developed in sac; patient lived 21 yrs. (No data as to sacral defect. Case noted briefly by Flickenger & Masson).
22	Judd (1915)	F	38	Unknown	Posterior approach	Cured	Case noted briefly by Flickenger & Masson
23	Coqui (1916)	F	20	Dystocia	Laparotomy with drainage	Cured	
24	Roux (1918)	M	8	Constipation; dysuria	Laparotomy; aspiration	Cured	
25	Frazier (1918)	F	52	Constipation; dysuria	None	Unknown	X-ray defect of S1 and S2
26	Weibel (1920)	F	47	Cyst of ovary (?)	Laparotomy with excision	Died; meningitis	Anterior and posterior defect of sacrum

TABLE I.—*Continued*

Case No.	Author (Date)	Sex	Age (Yrs.)	Symptoms	Treatment	Result	Remarks
27	Weber (1921)	F	27	Dysmenorrhea; cyst of ovary	Laparotomy; drainage per vagina	Cured; meningitis & fistula	Double uterus and vagina
28	Kennedy (1926)	F	22 mos.	Rectal polyp (?)	Excision, polyp (?)	Died; meningitis	Anterior sacral and rectal meningocele with sacral defect
29	Sabatini (1927)	F	3 mos.	Posterior meningocele	Excision, sac (posterior)	Died; etiology (?)	Anterior and posterior meningocele; teratoma
30	Demel (1928)	F	(?)	Gluteal tumor	Excision, posterior approach	Cured	Pedicle through the greater sciatic foramen, therefore presented as gluteal tumor
31	Drennan (1929)	F	16±	Dysmenorrhea	Excision, posterior approach	Died; meningitis	Sacral defect demonstrated on postmortem examination
32	Pick (1929)	M	32	Pelvic abscess (?)	Aspiration per rectum	Died; meningitis	X-ray and clinical evidence of sacral defect
33	Luth (1937)	F	15	Appendectomy	Found during appendectomy; partial excision and rupture	Died; meningitis	X-ray defect of sacrum
34	Santy (1938)	F	(?)	Constipation; leg pain	Excision, posterior route	Cured; fistula for 6 mos.	X-ray defect of sacrum. Nerve tissue and dermoid in sac
35	Adson (1938)	F	22	Constipation; backache	Excision, posterior route	Cured	X-ray defect of sacrum; filum terminale in sac
36	Holt-McIntosh (1940)	?	Infant	Constipation	None; accidental rupture	Died; meningitis	Defect of sacral bodies
37	Coller-Jackson (1943)	F	22	Constipation	Excision, posterior route	Cured	X-ray defect of sacrum

38	Shidler-Richards (1943)	F	50	Pain, back and legs	Aspiration, transsacral	Died; septicemia	X-ray defect of sacrum
39	idem	M	29	Chronic ulcer, right foot	None	Unknown	X-ray defect of sacrum; anterior and posterior meningocele
40	idem	F	29	Constipation	Excision, posterior route	Cured	X-ray defect of sacrum; case proved by myelography
41	Ingraham-Hamlin (1943)	M	5 mos.	Constipation; dysuria	Laparotomy; reoperated upon with partial excision	Died; postoperatively	Glial tissue in sac. X-ray showed displacement of bowel; not stated if sacral defect; reproduction too poor to read
42	Brown-Powell (1945)	F	26	Sacral pain, dysuria, dystocia	Excision, posterior route	Cured	X-ray defect of sacrum. Case proved by myelography; sac showed dermoid characteristics and sympathetic nerve cells.
43	Flickenger-Masson (1946)	F	51	Pelvic tumor	Laparotomy; excision	Cured	X-ray defect of sacrum
44	idem	F	31	Rectal fistula, 4 yrs.	None	Died; meningitis	Not a reported case but briefly noted by these authors
45	Alexander-Stevenson (1946)	F	2 mos.	Tumor, right gluteal	Radiation therapy	Died; uremia	No sacral defect. Posterior meningocele presented thru greater sciatic foramen into the pelvis; teratoma in sac.
46	Eder (1948)	F	27	Dysmenorrhea, constipation; pelvic mass	Laparotomy; partial excision	Recovered; after meningitis	X-ray defect of sacrum; case proved by myelography; double uterus and vagina
47	Sherman et al. (1950)	F	55	Low backache; stress incontinence; 3 uneventful vaginal deliveries	Exposed via posterior route; aspirated and closed	No further symptoms	X-ray defect of sacrum. Presenting symptoms due to unrelated cystocele and rectocele
48	idem	F	32	No symptoms	None		No x-ray defect; retrorectal mass palpated; questionable case
49	idem	F	40	Constipation; retrorectal cyst	None		X-ray defect of sacrum; no myelogram

TABLE I.—*Concluded*

Case No.	Author (Date)	Sex	Age (Yrs.)	Symptoms	Treatment	Result	Remarks
50	Calihan (1952)	F	24	Headache associated with bowel movements	None		X-ray defect of sacrum; case proved by myelography
51	idem	M	7	Constipation since birth	None		X-ray defect of sacrum; displacement of rectum by barium enema; case proved by myelography
52	idem	F	8	Abdominal pain, pyuria	None		X-ray defect of sacrum; myelogram not done
53	Leigh-Rogers (1954)	F	18	Low back pain; constipation; recurrent pyelitis	Removal via anterior route; pedicle ligated	Cured	X-ray defect of sacrum
54	idem	F	48	Retrorectal mass discovered on gynecologic examination; no symptoms	Removed via anterior approach	Cured	X-ray defect of sacrum; febrile 14 days postoperatively; sac composed of fibrous connective tissue, muscle tissue, and myelinated nerve fibers; double kidney and ureters bilaterally
55	idem	F	45	Frequency and dysuria	None		X-ray defect of sacrum; case proved by myelography
56	idem	F	22	Frequency, dysuria and fever—all intermittent	None		X-ray defect of sacrum
57	Silvis et al. (1954)	M	32	Obstruction, sigmoid; headache; dysuria	1. Sigmoid colostomy; 2. Ligation of pedicle and aspiration of cyst via posterior approach; 3. Closure of colostomy and evacuation of hematoma	Cured	X-ray defect of sacrum; case proved by myelography; displacement and compression of colon by barium enema; displacement of ureters on I.V.P.

The abnormal observations on physical examination were limited to the abdomen and rectum. The former was distended, and peristaltic sounds were hyperactive. Diffuse lower abdominal tenderness existed, but muscular rigidity and rebound tenderness were absent. A large ovoid, smooth mass, which apparently arose in the pelvis inasmuch as it was not attached to the anterior abdominal wall and was not movable, filled the lower abdomen. Rectal examination revealed a large, tense mass which bulged into the right posterior aspect of the rectum. The coccyx and sacrum were not palpable because of this mass. Neurologic examination was negative. Routine and other indicated laboratory studies were normal.

A roentgenographic examination of the abdomen, following catheterization of the urinary bladder, revealed a moderately distended large bowel packed with fecal material; a rounded homogeneous shadow with the approximate density of water, located in the pelvis; and a deformity of the sacrum and coccyx that was typical of a scimitar sacrum (fig. 1). An intravenous pyelogram proved there was satisfactory renal function without evidence of obstruction, but both ureters were deviated laterally and anteriorly as they crossed the brim of the pelvis (fig. 2). Sigmoidoscopy disclosed a rectal ampulla that was sharply angulated anteriorly and to the left by an extraluminal mass.

A diagnosis of presacral meningocele was made, and was confirmed by myelography which demonstrated the migration of the radiopaque substance from the subarachnoid space into the pelvic mass (fig. 3).

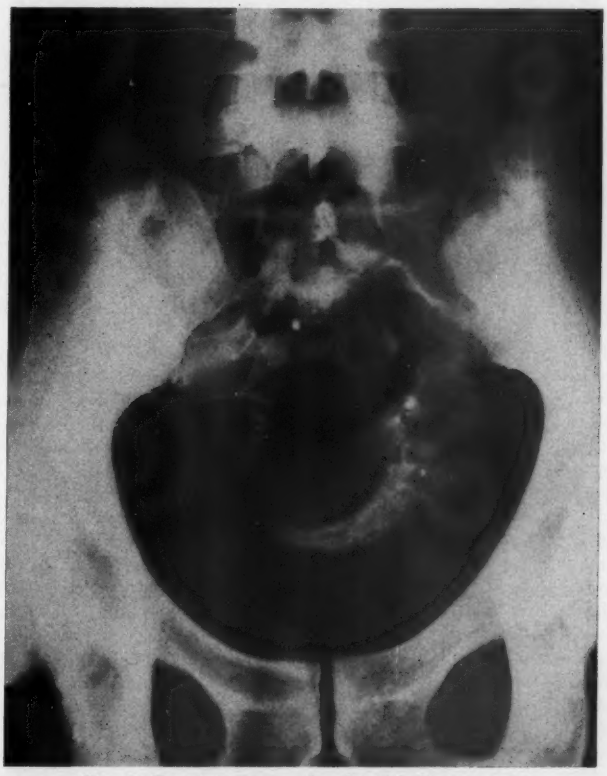

FIG. 1. Roentgenogram showing scimitar sacrum

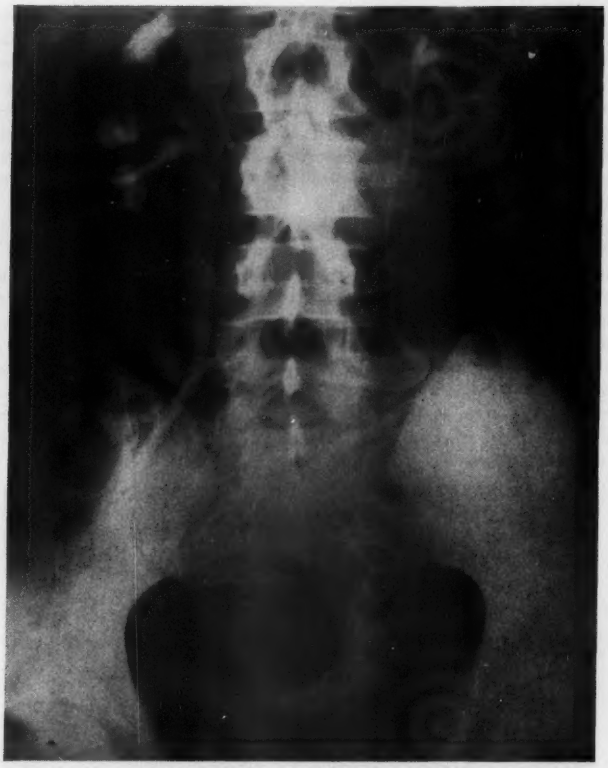

FIG. 2. Intravenous pyelogram showing lateral deviation of ureters in pelvis

Vigorous attempts to disimpact the colon by conservative measures were unsuccessful. While a colostomy was not required as an emergency measure, it was elected to perform a diversion colostomy for reasons that will be brought out in the discussion. Consequently, on the fifth day after admission, a diversion sigmoid colostomy, of the Wangensteen type, was performed through a left lower abdominal muscle-splitting incision. Palpation of the meningocele through this small incision was interesting, in that it was vastly larger than suspected and bulged high above the brim of the false pelvis.

Definitive Operation: On June 22, 1954, after thorough decompression, cleansing, and sterilization of the large bowel and rectum, the meningocele was attacked from the sacral approach 13 days after the colostomy. Due to the congenital absence of the left half of the sacrum, the exposure of the meningocele was simple and almost bloodless (fig. 4). The meningocele was opened and 1075 ml. of clear fluid aspirated (fig. 5). The posterior opening was enlarged and the interior inspected with the aid of a lighted retractor (fig. 6). The lining was smooth, glistening, and contained no visible nerve tissue. The communication with the subarachnoid space, superiorly, was identified. The smallest available probe (1 mm.) could not be passed through it, yet drops of cerebrospinal fluid were noted to emerge at intervals.

The neck of the meningocele was mobilized and divided. A pledget of gelfoam was placed over the opening, following which the dura was closed with interrupted no. 0000 silk sutures.

Oozing from the anterior portion of the divided sac was controlled with a lock stitch of fine catgut. No attempt was made to remove the large, collapsed, isolated wall of the meningocele, and the incision was closed in layers without drainage.

Postoperatively, the patient was kept on a Bradford frame with his hips above his head. Penicillin, streptomycin and sulfadiazine were administered. On the third day following repair of the meningocele marked pallor appeared, and hemorrhage was evident. The erythrocyte count was 2,300,000 per cu. mm.; the hemoglobin, 7 Gm. per 100 cc., and the hematocrit, 25. Rectal examination revealed a large boggy mass in the area of the former meningocele. It therefore was clinically apparent that the patient was suffering blood loss into the meningocele sac. The approach to this sack through the lower abdominal wall would be fraught with peril because of the colostomy and the fact that the patient was but 3 days' postoperative. Therefore, conservative measures, consisting of the administration of 1,500 ml. of whole blood, were adopted and within the next 48 hours the blood count had returned to normal. There was no subsequent evidence of bleeding.

The posterior sacral operative wound healed *per primam*. There was no discharge of meningeal fluid and the patient exhibited no signs of meningeal irritation at any time during his convalescence. A low grade fever and malaise, incident to the hemorrhage within the meningocele sac, persisted until the time of the final operation.

On Aug. 6, 1954, 44 days after the repair of the meningocele, and after extensive steriliza-

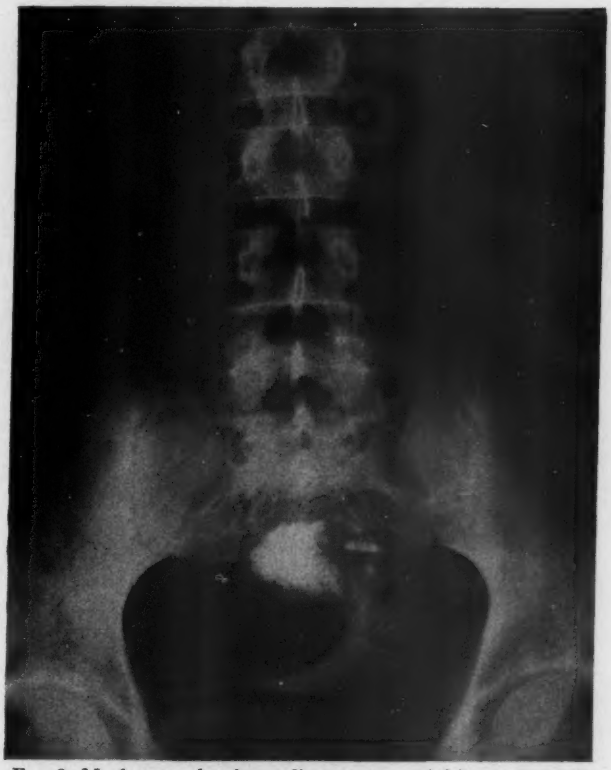

FIG. 3. Myelogram showing radiopaque material in meningocele

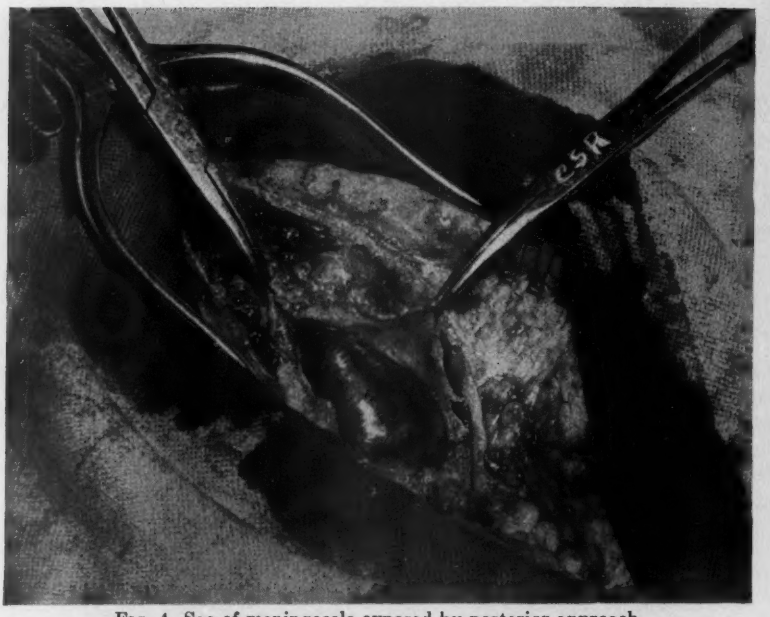

FIG. 4. Sac of meningocele exposed by posterior approach

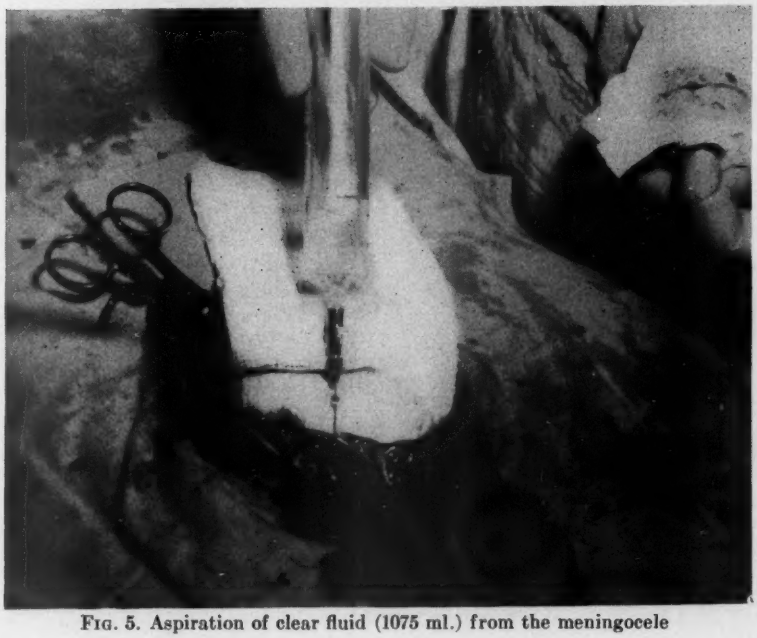

FIG. 5. Aspiration of clear fluid (1075 ml.) from the meningocele

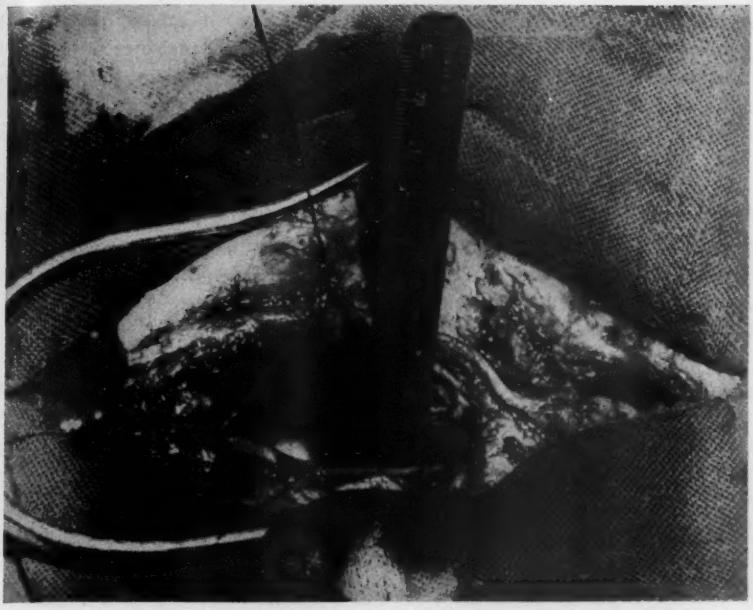

FIG. 6. Interior of meningocele sac

tion of the colon, the sigmoid colostomy was closed. After a triple preparation of the abdomen with Phisohex® and a change of drapes, gloves and instruments, the meningocele sac was exposed through a right lower abdominal quadrant, extraperitoneal, muscle-splitting incision. Approximately 900 ml. of dark clotted blood was evacuated from the former meningocele cavity. A drain was inserted. The postoperative course was uneventful, and both incisions healed *per primam* within a week.

Follow-up examinations have disclosed that the patient has had no headaches, has had normal stools once to twice daily without laxatives, and has been entirely symptom free for a period of more than 1 year.

DISCUSSION

In view of the misadventures in the past, it would appear that definitive correction of anterior meningoceles is warranted upon diagnosis. Any decision is to be tempered with surgical judgment, but the majority of such meningoceles that are diagnosed will be comparatively large, and may be subject to disaster in the future.

The posterior approach for the definitive surgical treatment is strongly preferred.[2] The most frequent cause of death in the patients operated upon has been meningitis. Despite meticulous technic, drainage of spinal fluid may result, for a time, from the sacral wound. A well-known surgical principle is that "*whenever fluid drains out, some fluid seeps back in.*" Therefore, it appears to us that since the mortality rate to date in the surgical correction of this abnormality has been so high, elective diversion sigmoid colostomy is justifiable. If a tempo-

rary spinal fluid leak occurs, a defunctionalized anus will greatly decrease the incidence of meningitis. And, of great importance, if the presence of a complicating teratoma indicates partial or total extirpation of the sac, the accidental entering of the lumen of a tightly adherent, scarred sigmoid or rectum is a hazard that would be much less perilous if the lower bowel were defunctionalized.

SUMMARY AND CONCLUSIONS

The congenital anomaly, anterior sacral meningocele, is discussed with particular reference to an explanation of its actual frequency.

Fifty-six cases are tabulated, and 1 case is added to the literature.

Diversion sigmoid colostomy, complete bowel "*sterilization*" and the posterior sacral approach for the definitive surgery are advocated.

U. S. Naval Hospital
Oakland 14, Calif.

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MEDIASTINAL EMPHYSEMA AND BILATERAL PNEUMOTHORAX FOLLOWING SURGERY OF THE NECK

LUTHER C. ROLLINS, M.D., DAVID HENRY POER, M.D.*

Atlanta, Ga.

Mediastinal emphysema is an authentic, proved complication of neck surgery. In 1918 Buford[3] first discussed this condition following operations other than tracheotomy. In 1934 Keis[5] reported a case of mediastinal emphysema and pneumothorax following thyroidectomy and mentioned 7 cases reported in the literature. Seed[8], in 1949, described 2 cases of thyroidectomy with this complication and collected from the literature 16 cases with death in 11 patients.

This complication has been reported after tracheotomy, radical neck dissection, stellate block, stellate ganglionectomy, esophageal surgery, tonsillectomy, and anesthesia alone. Lahey and Warren[6] did not report this condition as a complication in 365 cases of esophageal diverticula.

Macklin[7] produced experimental mediastinal emphysema and pneumothorax on cats and other animals in 1939. This was done using closed pressure symptoms with forced air. Hyperinflation of the intrapulmonary alveoli resulted in rupture and dissection of the air along the vascular sheaths into the mediastinum. The fragile mediastinal pleura ruptured to give a bilateral, ipsilateral, or contralateral pneumothorax while the visceral pleura remained intact. It was impossible to precipitate mediastinal emphysema by injection of air directly into the pleural cavity, even under high pressure.

Goldberg, Mitchell, and Angrist[4], on injecting air into the deep cervical fascial layers of 9 cadavers, 1 year of age or less, found that air ruptured into the pleural cavity at 40 mm. of Hg. Once ruptured, further ingress of air entered the pleural cavity at only 0.5 mm. Hg. This finding readily explains the high mortality rate of this disturbance, namely, tension pneumothorax, rapidly developing and undiagnosed.

In 5 cases terminating fatally due to this complication, the pleura was intact at autopsy and no interstitial emphysema arising from hyperinflation could be demonstrated.

REPORT OF CASE

Mrs. J. G. M., a 63 year old housewife, was admitted to the hospital on Feb. 7, 1954, complaining of "*wanting to swallow all the time.*" She first noticed this difficulty 6 months prior to admission, at which time she became conscious of the persistent desire to swallow something low in her throat. There was no associated dysphagia, but she had experienced occasional postprandial eructations and the regurgitation of old food. Her appetite had remained good, and she reported no loss in weight.

Two weeks prior to admission, Dr. Murdock S. Equen had demonstrated an esophageal diverticulum of the pulsion type about 1.5 by 2 cm. in size on the left side.

* Departments of Surgery, Piedmont Hospital and Emory University School of Medicine, Atlanta, Ga.

The history was not remarkable, and on physical examination no cervical masses or tenderness, or other significant findings were noted.

On February 8, with a nasogastric tube in place and under endotracheal anesthesia, an oblique incision of the neck was made on the left along the anterior border of the sternocleidomastoid muscle. A diverticulum of the pulsion type was found posterior and inferior to the inferior pharyngeal constriction muscle. Excision of the sac and high ligation were feasible in one stage, and no anesthetic or surgical complications occurred during the entire procedure, which lasted 2 hours. There was no evidence of pleural injury on the left at the time of surgery, and no sucking noises or hissing sounds were heard during the procedure.

Postoperatively, the patient reacted normally, but late in the afternoon began to complain of pain in the chest, substernal at its incipience but progressing to a pleuritic type of pain. The heart and breath sounds were distant, and the point of maximal impulse was not located; however, no subcutaneous emphysema or mediastinal crunch was noticed. On the following morning, a roentgen study of the chest demonstrated bilateral pneumothorax, 50 per cent on the left and 20 per cent on the right (fig. 1).

At this time the blood pressure was 135/80; the pulse was regular, the rate varying from 90 to 100; respirations ranged from 20–24, and the temperature varied from 99 to 100 F. As vital signs were not alarming, a conservative regimen was followed, which included

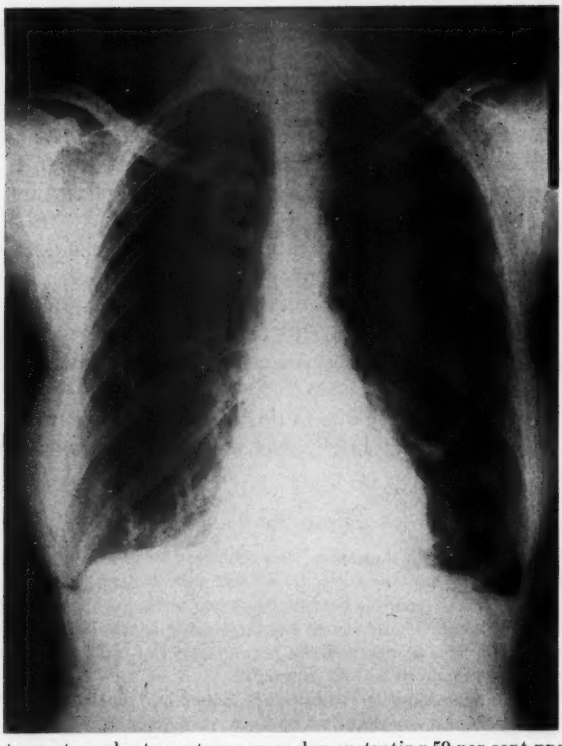

FIG. 1. Posteroantero chest roentgenogram demonstrating 50 per cent pneumothorax on the left, 20 per cent pneumothorax on the right. First day postoperative.

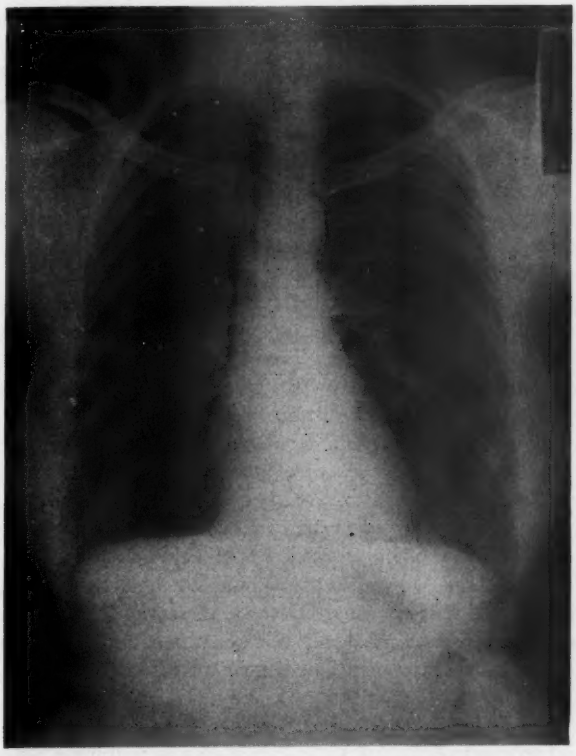

FIG. 2. Posteroantero chest roentgenogram demonstrating complete re-expansion. Twenty-one months postoperative.

close observation, antibiotics, bed rest and feedings by nasogastric tube. At no time was there cyanosis or acute respiratory distress, or evidence of a tension pneumothorax.

By February 10, subcutaneous emphysema was pronounced, but on February 12 roentgen examination of the chest revealed no progression of the pneumothorax.

Subsequently the postoperative course was uneventful. The Levin tube was removed on the eighth postoperative day. The patient enjoys excellent health at present, 2 years later. A chest roentgenogram in November 1955 reveals full re-expansion of both lungs (fig. 2).

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

The cervical fascia is comprised of superficial and deep layers. The deep cervical fascia is divided into superficial, pretracheal, and prevertebral layers. It is the pretracheal fascia which is of concern here, as this middle layer of the deep fascia extends inferiorly into the mediastinum, where it ultimately blends with the fibrous pericardium. The prevertebral or deep layer is continuous inferiorly with the endothoracic fascia. Between these layers, a free passageway into the

mediastinum is provided by opening the middle layer of the deep cervical fascia.

Thus, any surgery of the neck which involves the opening of the pretracheal fascia is fraught with the possible complication of mediastinal emphysema and pneumothorax. It is explained, not by the fact that the pleura at the apex of the lung is injured, but by the fact that air enters the mediastinum through the suprathoracic aperture. This happens during inspiration when there is an increased negative intrapleural pressure, as in coughing and laryngospasm, thus aggravating the already increased intramediastinal pressure.

Air under tension in the mediastinum may rupture the fragile mediastinal pleura, producing an ipsilateral, contralateral, or bilateral pneumothorax. This air may dissect upward into the neck, face, axilla, or thoracic wall or follow the aorta and esophagus into the retroperitoneal area. Indeed, air traverses fascial planes readily under slight pressure. Recently, a pneumopericardium was observed in this hospital following a diagnostic Rubin test for sterility.

The alternate explanation for the development of this condition is a weakening of the marginal perivascular alveolar bases with escape of air from the alveoli into the perivascular sheaths. This would be the fault of the anesthesiologist or anesthetist when using a closed system exceeding safe intra-alveolar pressure. If pressure of 15 mm. Hg is not exceeded in the closed system, this mechanism of the production of this clinical entity should not be a factor. Should air gain access to the perivascular sheaths, however, dissection along these structures into the mediastinum is rapid, and with rising intramediastinal pressure, pneumothorax occurs as in the aforementioned mechanism.

DIAGNOSIS

There are five cardinal factors to be considered in making a diagnosis of this condition. They are:

1. Subcutaneous Emphysema. Probably the most reliable sign in the immediate postoperative period is subcutaneous emphysema, which usually is present over the anterior and lateral wall of the chest, the axillas, shoulders and neck. It occurs in more than half of the patients.

2. Pain in the Chest. The pain may be substernal initially, suggesting myocardial infarct, or it may be more commonly pleuritic and lateral.

3. Variation from Normal Respiratory Patterns. There may occur (1) tachypnea with or without dyspnea, and orthopnea with progressively decreasing vital capacity in case of a developing tension pneumothorax; (2) the diaphragmatic or abdominal type of labored breathing; (3) the prolonged expiratory phase; and (4) cyanosis of varying degree, an indication for immediate needle aspiration or closed thoracotomy.

4. Evidence of Circulatory Failure. (1) Shock with cool moist skin, tachycardia and hypotension may indicate circulatory failure, but following restoration of normal respiratory physiology it frequently will correct itself. Blood transfusion may be indicated, but only after the disturbed respiratory physiology has been corrected. (2) On palpation, there may be evidence of splinting

of the corresponding side of the chest to the pneumothorax. (3) Hyperresonance may be noted on percussion.

5. Auscultation. Distant breath sounds and heart sounds may be detected by auscultation, and there may be a mediastinal crunch (Hamman's sign) due to air in the mediastinum.

TREATMENT

One must be conscious of the possibility of this complication and aware of the correct measures to restore normal respiratory physiology. Positive pressure breathing in cyanotic, apneic patients with this disturbance is definitely contraindicated. While indicated with open pneumothorax in reinflating the collapsed lung, it only magnifies respiratory embarrassment in any case of tension pneumothorax. This is particularly true in patients when the precipitating factor has been rupture of marginal alveoli.

The importance of prompt and adequate therapy cannot be over-emphasized. An airway is essential, if the patient has not yet reacted, along with oxygen, either nasally or by tent, but not by positive pressure mask system. Analgesics, and demerol rather than morphine since the latter depresses respirations, are used to alleviate anxiety as well as for beneficial effects on shock and pain. Close observation is mandatory as the development of tension pneumothorax merits immediate, life-saving needle aspiration and/or closed thoracotomy drainage of the third or fourth intercostal space.

Should there be little change in the vital signs and if the status of the patient is reasonably satisfactory, it is justifiable to wait for roentgenologic confirmation. Lateral views are valuable in demonstrating the mediastinal and subcutaneous air.

SUMMARY

Mediastinal emphysema and pneumothorax constitute a severe although uncommon complication of any surgery of the neck involving incision into the pretracheal or middle layer of the deep cervical fascia.

The surgeon may cause this complication if the pleura is inadvertently incised beneath Sibson's fascia at the cupola of the lung and the anesthesiologist may be at fault if hyperinflation of the pulmonary circuit has occurred. In many instances, however, the air enters the mediastinum and pleural space directly by way of fascial planes, as has been confirmed by autopsy findings.

The knowledge of the possible development of this clinical entity and the ability to recognize its presence are essential to diagnosis.

The most common early findings are subcutaneous emphysema, pain in the chest, variation from the normal respiratory pattern, evidence of circulatory failure, and auscultatory findings. Roentgen examination of the chest corroborates the diagnosis and may be used to follow the progress of this complication.

Oxygen, analgesics, and the use of an airway in the unreacted patient are discussed. The instigation of immediate needle aspiration or closed thoracotomy

drainage in tension pneumothorax is mandatory and positive pressure apparatus should be avoided.

Close observation is essential to lower the high mortality rate of this unusual postoperative complication.

Piedmont Hospital
Atlanta, Ga.

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GIANT CYST OF THE KIDNEY*

A CASE REPORT

HOWARD JACOBS, M.D., HARRY C. SALTZSTEIN, M.D.

Detroit, Mich.

As a clinical entity, serous cysts of the kidney are not as rare as they were formerly thought to be.

Renal cysts were described in literature as early as 1650 by Fabricus of Hilden, known as the father of German surgery. Many isolated reports in the early literature referred to simple cysts of the kidney, but it was not until the early twentieth century that any comprehensive research was done on the subject. In terms of percentage, it has been stated that large renal cysts are seen in from 3–5 per cent of routine autopsies.

In general, renal cysts can be divided into three major classifications[6]: (1) The minute cysts of kidney. This condition is not a clinical one. These cysts may be found incidental to a genitourinary workup; they are thought to be due to small structural defects. (2) There is the polycystic kidney and (3) the large solitary cysts.

The following case, involving a giant solitary cyst, is unusual because of its size and relative absence of symptoms.

CASE REPORT

The patient, a 16 year old white female, was admitted to Sinai Hospital, Detroit on March 12, 1955. She was perfectly well until 3 days prior to admission at which time she suddenly awoke at 3:00 a.m. with severe nausea, and after a large emesis, felt well and went back to sleep. There was no pain or distress. The following morning she ate breakfast, but soon after noted gradual and progressive pain in the right lower abdomen. It was *"gnawing"* and persistent, aggravated by movement and did not radiate. This persisted for 2 days and was accompanied by mild nausea and low grade temperature (99.6–99 F.).

Her menses had been regular. Her last menstrual period was normal and occurred 15 to 17 days prior to admission to the hospital. She had had no previous abdominal or urinary symptoms whatever. There had been no previous surgery.

She was admitted with the diagnosis of acute appendicitis. Physical examination was negative except for the abdomen. There was moderate tenderness to deep palpation in the right lower and mid quadrants with some rebound. It was not urgent, and observation was decided upon. The next day, careful examination revealed a remarkable finding in the abdomen. There was asymetry. Filling the entire left side of the abdomen, and extending to the right, 1 or 2 inches below the umbilicus was a nontender, firm mass, dull to percussion and eliciting a fluid wave (fig. 1). Bowel sounds were normal. The white blood cell count was 8,600 per cu. mm., the hemoglobin was 15.4 Gm. per 100 cc. The urine had 5–6 white blood cells per high power field. All other laboratory studies, including repeated urinalyses, were negative.

The radiologic studies were most impressive. Flat plate of the abdomen (fig. 2) revealed a large homogeneous density present in the left upper quadrant and extending to below the umbilicus and below the crest of the left ilium. Further intravenous pyelography

* From the Surgical Service of Sinai Hospital, Detroit, Michigan.

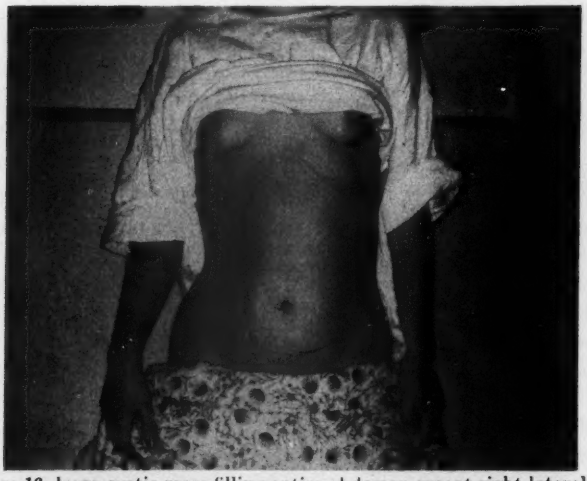

FIG. 1. Age 16, large cystic mass filling entire abdomen except right lateral area. Three day history of nausea and abdominal distress.

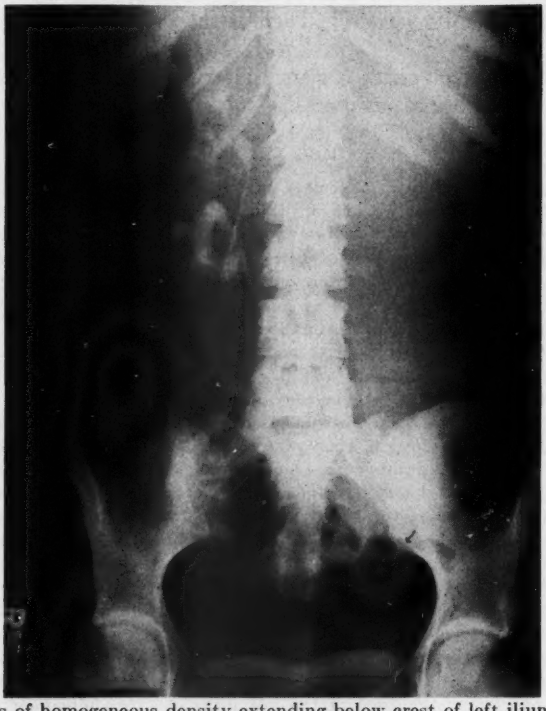

FIG. 2. Mass of homogeneous density extending below crest of left ilium. Intravenous pyelography shows right kidney of normal size and position. Left kidney is completely transposed to the right side of the abdomen, lying below the right kidney, and is somewhat rotated. Left renal pelvis is normal in diameter. Left ureter is seen returning to left side of abdomen and in the pelvis, it is normal in position (arrow).

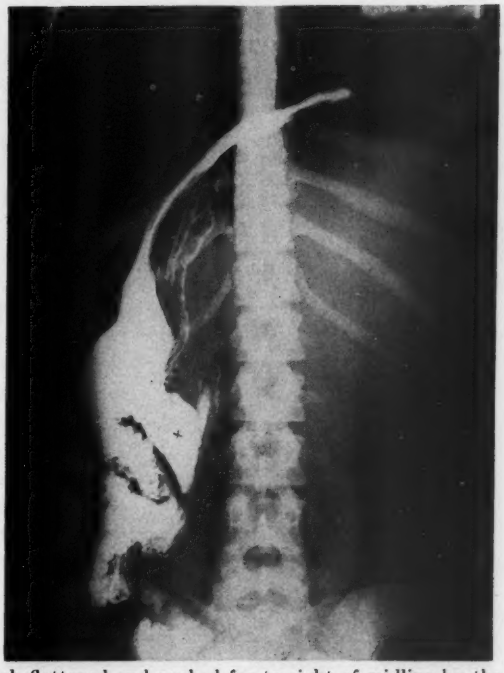

FIG. 3. Stomach flattened and pushed far to right of midline by the mass. The third portion of the duodenum and the jejunum distal to ligament of Treitz (×) elevated and displaced to the right side of the abdomen.

showed the right kidney to be normal in size, shape and position. The left kidney was deviated completely to the right of the midline and somewhat rotated and was lying below the right kidney. The left renal pelvis was normal in diameter and the left ureter could be seen returning to the left side of the abdomen, and in the pelvis it was normal in size and position.

An upper gastrointestinal series (fig. 3) showed the stomach to be flattened by the mass and shifted to the right of the midline. Abdominal aortography and retroperitoneal air insufflation (fig. 4) showed that the aorta had shifted to the right of the midline. The mass was sharply demarcated by the air, indicating its retroperitoneal nature.

Nine days after admission, exploratory laparotomy was carried out through a long transverse upper abdominal incision. "An enormous cyst presented itself, filling the entire upper abdomen down to the sacrum. A thin covering over it was divided, which proved to be the posterior peritoneum. The cyst could now be further explored. It measured over 15 inches in diameter and was attached to the superior pole of the left kidney. It extended high up beneath the diaphragm and downward to below the bifurcation of the aorta. It was not deeply attached, and the wall was rather firm. It was attached to the left kidney, which had been displaced over to the right side of the abdomen.

"The cyst was dissected away from the left kidney. It communicated with the pelvis of the kidney. A small rent was made in the cyst wall near the kidney pelvis, but then the cyst was easily lifted out of the abdomen. The left adrenal gland was plastered against the cyst; this was easily dissected away and preserved. The cyst measured fully 15 inches in diameter. It contained approximately 5 liters of thin, rather dull colored watery fluid."

"The left kidney was now reconstructed by closing the area where the cyst had been

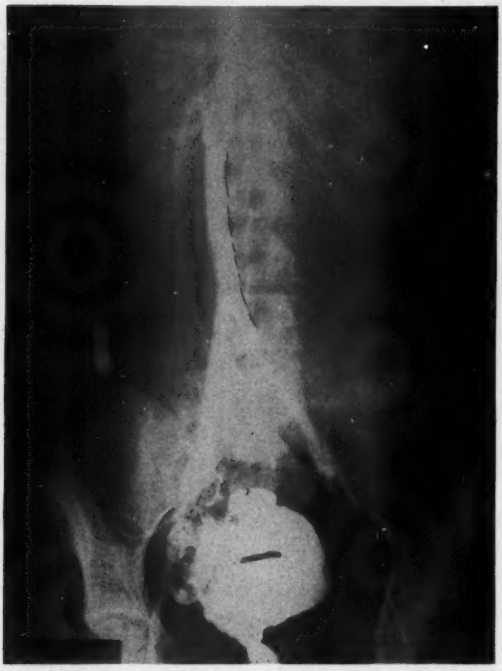

FIG. 4. Abdominal aortography. Aorta shifted to the right of the midline

with several sutures, and was then easily replaced into its normal bed in the left flank. A catheter was placed in the pelvis extending down the ureter, the other end brought out through the posterior kidney parenchyma, and lead out through a small incision in the left lumbar abdominal wall below the twelfth rib."

Convalescence was uneventful except for some drainage for 3 to 4 weeks from the raw retroperitoneal surfaces in the left subdiaphragmatic region. Microscopic examination showed the cyst wall lined with low cuboidal cells. There were fibrous connective tissue and remnants of glomerulae and atrophic tubules in it. The renal tissue opposed to the surface showed moderate interstitial fibrosis. There was no evidence of malignancy.

DISCUSSION

Solitary cysts of the kidney are usually found at the lower pole; however they may occur anywhere. The largest cyst we were able to find mentioned in the literature, contained 12 liters of a serous fluid.[4] Although the size of these cysts varies considerably, the majority usually measure between 5 to 10 centimeters.

The exact cause of simple serous cysts of the kidney is not known. It appears that they can be congenital or acquired. It is the opinion of Herbut[7], that most, if not all of these cysts, are congenital in origin; the same theoretic considerations are applicable in these lesions as in polycystic disease. Hepler,[7] on the other hand, has produced large solitary cysts in rabbits by fulguration of the papilla and ligation of the posterior branch of the renal artery. Thus, tubular obstruction

and atrophy of the kidney parenchyma are probably etiologic factors in acquired simple cysts. The size of a cyst and the direction of its enlargement in the abdomen are factors causing the symptoms which it may produce. It may be heralded by a variety of symptoms referable to other systems, due to its pressure against neighboring organs. On the other hand, a patient may live an entire life span with complete absence of symptoms.[4] If the cyst is of sufficient size, it usually can be palpated through the abdominal wall. However, on physical examination, with a mass presenting itself in the left upper quadrant, one must also consider the possibility of enlarged spleen, cysts of the tail of the pancreas, omental cysts and retroperitoneal neoplasms.[2] Diagnosis will depend largely on radiographic findings.

Braasch,[1] in a review of 149 simple cysts of the kidney, described the urographic abnormalities as follows: The most typical is a crescentic outline of the border of the adjacent calyx. Other deformities of the calyces, in order of frequency, are "elongation, dilatation, displacement, general deformity and flattening of the border of the pelvis adjacent to the cyst." In 14 of the 149 patients, there was no deformity in the urogram. This occurs when the cyst is far enough away from the renal pelvis so that there is no pressure on it, and is more frequent in cysts of the lower pole and also with subcapsular cysts. "The greatest degree of deformity is observed when a large cyst is situated in the central part of the kidney."

Sometimes a flat plate suggests the outline of the cyst. It is seen as a round, dense shadow adjacent to, or overlying the kidney. The outline of a cyst is more circular and regular than that of a neoplasm, and often but not always, the fluid in the cyst casts a denser shadow than does the neoplastic tissue. The deformity of the renal pelvis is greater with a neoplasm than with a cyst. Even with all the clinical data available, a diagnosis still may not be made until the patient undergoes surgery.

The treatment of large serous cysts of the kidney, or even small cysts which produce symptoms is surgery. It may be difficult at times to ascertain from pyelographic findings alone if one is dealing with a simple cyst, a neoplasm, or a cyst associated with neoplasia.[5] One author has quoted a 25 per cent incidence of carcinoma associated with hemorrhagic cysts of the kidney. Aspiration of cyst contents with injection of 50 per cent glucose has been reported.[3] It is the belief of most authors, however, that this method of therapy should be reserved only for those patients in whom surgical removal is impractical.

SUMMARY

A large serous cyst of the kidney is reported. The cyst was 15 inches in diameter. It arose from the left kidney, which had been pushed far over to the right side and above the right kidney.

The cyst was resected, and the left kidney replaced into its normal position.

966 Fisher Bldg.
Detroit 2, Mich.

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TRAUMATIC RUPTURE OF THE LIVER WITH SEVERE SECONDARY HEMORRHAGE FOLLOWING REMOVAL OF A GAUZE PACK*

I. Arnold Jaffe, M.D.

Bronx, New York

Deaver and Ashurst[2] attribute the first operation for hemorrhage from a liver wound to Burckhardt, who in 1887, packed a wound of the liver. In 1896 Kousnetzoff and Penski[3] described several methods of suture of liver wounds. At the turn of the century the surgeons main concern in liver wounds was the control of hemorrhage. Many ingenious methods were devised for the control of bleeding. At this time the operative treatment for wounds of the liver became more common. In 1912 Thole[6] collected 680 reports of operative cases of liver wounds.

During World War I[1] the mortality rate for liver wounds was reported to have been 66.2 per cent. In World War II the operative mortality rate for wounds of this type was markedly reduced. During this period Lawrence, Madding and Kennedy[4] described 829 wounds of the liver with a mortality rate of 27.0 per cent. Vigorous treatment to combat shock, early surgery, the use of antibiotics and better treatment of other related injuries, account for the reduction in mortality.

No generalization as to the treatment of liver wounds can be made. There are varying opinions regarding the exact treatment and management of precise types of wounds of the liver.

The case described in this report is that of a rough fracture of the liver following a fall onto an iron fence, in which neither the skin nor rib was broken. It is believed that this case is worth reporting in order to point out that:

1. Massive tears of the liver instead of being considered hopeless, can be attacked with some reasonable measure of success.

2. The removal of a gauze pack used to stop profuse bleeding, may be accompanied by severe secondary hemorrhage.

CASE REPORT

J. W., a 20 year old white man, was admitted to the second surgical division of Fordham Hospital on June 12, 1954 in acute shock. The history as ascertained from the patient was that while leaning against a porch rail, the rail gave way. He fell about 10 feet onto a 4 foot high iron fence, hitting the corner of the fence against his right chest wall.

Physical examination revealed an acutely ill, thin white man in severe pain and in shock. There was a large bruise over the seventh right intercostal space at the level of the nipple line. The pulse was thready and rapid at 120 per minute and the blood pressure was 90/60. The hematocrit was 27 volume of packed cells. The abdomen was tense with marked tenderness and rigidity over the entire abdomen, and most marked in the right upper quadrant. Radiographic examination of the chest showed no evidence of rib fracture, with both lung fields clear. Roentgenologic examination of the abdomen showed no evidence of free air under the diaphragm. A diagnosis of a ruptured liver with intra-abdominal bleeding was made. It was decided that if signs of intra-abdominal bleeding continued

* From the second Surgical Division of Fordham Hospital, New York.

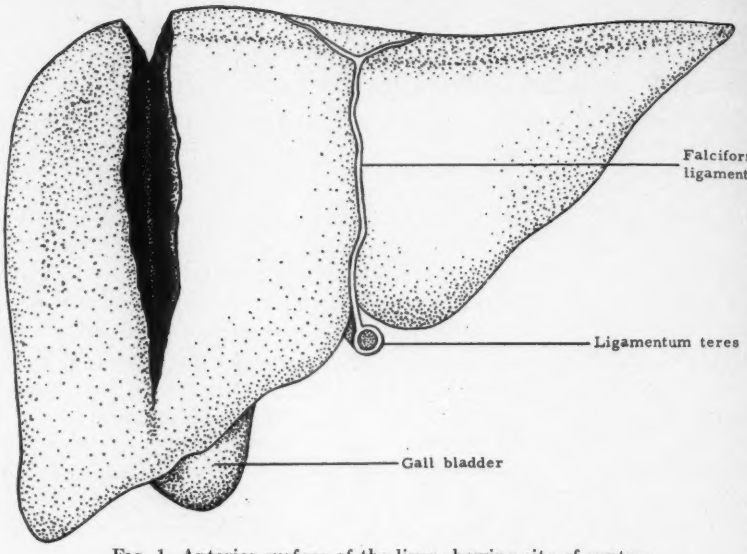

FIG. 1. Anterior surface of the liver showing site of rupture

exploration of the abdomen would be performed. In the meantime the patient was given 100 mg. of demerol and an infusion of 500 cc. of 5 per cent dextrose in water which was followed by 500 cc. of blood. Re-examination of the patient in 2 hours showed no improvement in his general condition nor of his abdomen. Exploration was decided upon at once.

The abdomen was entered by means of a right rectus muscle splitting incision. Handfuls of blood clot and a large quantity of fluid blood was evacuated from the abdomen. The incision was extended to the right. Examination of the liver revealed a large stellate rupture of the anterior surface of the right lobe extending from just above the inferior edge into the dome of the liver (fig. 1). This tear appeared to be about 5 inches long and extend into the depth of the liver about 3 inches. There was active bleeding from this area. Compression of the hepatic artery had little effect on the bleeding. At this point the pulse and blood pressure disappeared. Nitrous oxide was stopped and oxygen under positive pressure was given. Blood was forced into both the right and left arms under positive pressure. Attempts at suture of the liver were abandoned, and the rupture in the liver was tightly packed with a 4 inch leg roll type of gauze bandage. This appeared to stop the bleeding. The abdomen was closed with through and through silk sutures bringing the distal end of the bandage out of the lower edge of the incision. Fifteen hundred cc. of blood were given while the patient was in the operating room. His pulse improved and the blood pressure returned to 100/60, at the end of 40 minutes. Five hundred cc. of blood were given to the patient while he was on the ward. The patient recovered from this acute episode so that by the sixteenth of June his hemoglobin was 85 per cent, the hematocrit 44 per cent volume of packed cells, the pulse rate 80 per minute and the blood pressure was 120/80.

On the afternoon of June 16 the patient had a severe chill and a temperature of 105 degrees. This temperature persisted for 24 hours. It was thought that the chill might be due to the gauze pack. The patient was taken to the operating room, and the pack was removed by gentle traction on the end protruding through the lower end of the abdominal incision. Preparation for laparotomy was made in the event of severe bleeding. As the pack was removed, there was an escape of much serosanguinous fluid, but no fresh blood. The patient tolerated this procedure well. His abdomen remained soft, the pulse rate was 80 per minute and the blood pressure was 122/82 at the end of half an hour. Since there appeared

to be no evidence of fresh bleeding, the patient was removed from the operating room. While being put into bed, on the ward, the patient appeared to go into shock. This occurred 1 hour following the removal of the gauze pack. The pulse and blood pressure were imperceptible. His dressing became saturated with bright red blood, and the abdomen became tense and tender. Fifteen hundred cc. of blood were pumped by positive pressure into each arm. It was decided that if signs of active bleeding continued, exploration of the abdomen would be indicated. In about 1 hour the blood pressure was 96/48. At the end of 2 hours the pulse rate was 110 per minute and the blood pressure was 120/82. The abdomen appeared less tender and tense, and it was believed that active bleeding had stopped. Again the patient recovered from this acute episode.

He developed a biliary fistula which drained profusely for 1 month. A series of liver function tests on July 12, 1954 were as follows; cholesterol 185 mg., cholesterol esters 145 mg., total protein 7 Gm., albumin-globulin ratio 1.5:1, cephalin flocculation test negative, thymol turbidity 3 units, serum bilirubin total 0.4 mg. per 100 cc. The biliary drainage gradually decreased and the patient was discharged to the out patient department on August 4. The biliary fistula closed completely on Nov. 23, 1954. Examination on March 3, 1955 showed a healed abdominal scar with no evidence of herniation. He felt well and was employed as a truck driver. Liver function tests on that date were normal.

Comment: Madding, Lawrence and Kennedy[4] in a review of 829 war wounds of the liver showed the trend was away from the use of a gauze pack to control bleeding. Thirty-four and one-tenth per cent of their patients were treated by the use of a gauze pack in 1944, while in 1945 9.6 per cent were so treated. However, Sparkman and Fogelman[5] in a review of 100 consecutive cases of patients with liver injury who were admitted to Portland General Hospital at Dallas, Texas, felt no special method of therapy was applicable to all varieties of liver wounds. They used suture, drainage, packing and debridement, and combinations of the foregoing. They believed there were instances in which the character of the wound, the extent of the hemorrhage, and the condition of the patient dictate the use of a gauze pack as a life saving procedure.

Where a gauze pack has been used to control bleeding, Madding, Lawrence and Kennedy[6] advocate its early removal in the prevention of secondary hemorrhage. They believe early removal of the gauze pack promotes closure of external drainage. It is interesting to note that Madding[4] observed a fatal secondary hemorrhage on the eighteenth postoperative day following the removal of a gauze pack.

It would seem that the method of treatment in rupture of the liver will depend upon the character of the wound, the extent of the hemorrhage, and the condition of the patient. The removal of a gauze pack, used to control hemorrhage, will always be a hazardous procedure.

SUMMARY

A case of a ruptured liver with severe hemorrhage is described. A gauze pack was used to stop bleeding. The removal of the gauze pack was followed by a severe secondary hemorrhage.

The case described in this paper has been presented with the kind permission of Dr. A. B. Sullivan, director, Second Surgical Division, Fordham Hospital.

2539 Holland Ave.
Bronx, N. Y.

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FAMILIAL POLYPOSIS OF THE COLON

ROBERT P. MCBURNEY, M.D., ROBERT L. SANDERS, M.D.*

Memphis, Tenn.

Increasing experience with polyposis of the colon has established beyond doubt that colonic polyps are premalignant lesions and their presence constitutes a threat to the life of the patient. The treatment of the isolated polyp or even a few polyps is not too difficult; familial polyposis, however, wherein the colon contains literally thousands of the growths, frequently presents a serious surgical problem.

Fortunately, familial polyposis is relatively rare. Dukes[5] reviewed the evidence for the hereditary factor and reported 13 family pedigrees in which it developed. He showed that both sexes may be affected and may transmit the disease for several generations.

We are reporting the cases of 10 patients, all of whom had a family history of colon disease.

CASE REPORTS

Case 1. N. A., a white woman, 30 years of age, was first seen in May 1953. She gave a history of recurrent attacks of diarrhea and rectal bleeding over a period of 15 years. She had been treated for amebiasis, although amebas had never been found on stool tests. The family history revealed that many members of her family had had colon disease (chart I).

The only significant finding on physical examination was a small polyp in the lower rectum. Tests for amebiasis were negative. On proctoscopic examination, multiple polyps were found extending as high as 11 inches, and the roentgenogram demonstrated polyps throughout the colon (fig. 1).

The rectal polyps were fulgurated at the patient's initial visit. On July 27, 1953, a subtotal colectomy was performed and the ileum anastomosed end to end to the rectosigmoid at a level which could be easily visualized through the proctoscope. The patient's convalescence was uneventful and she was dismissed from the hospital 2 weeks postoperatively.

Pathologic examination of the specimen revealed a polyposis involving the entire colon (fig. 2). Three of the growths contained a low grade carcinoma with early invasion of the stalks. No metastases were found in the 32 lymph nodes which were removed.

This patient has been observed on several occasions since the operation, and on each of 3 visits 2 small polyps were fulgurated. At her last visit, Dec. 14, 1955, the area from the anastomosis to the anus was free of disease.

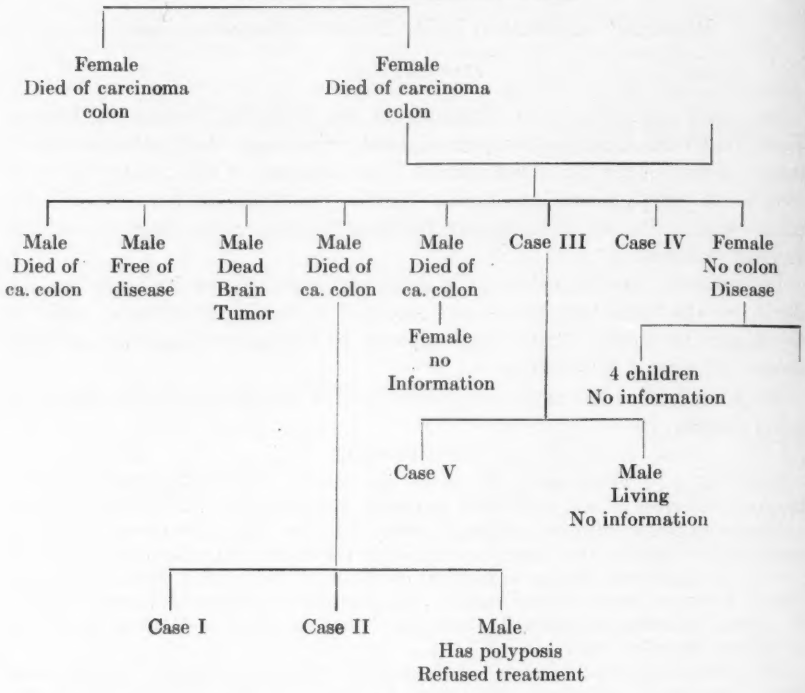
Case 2. A. M. E., a sister of the first patient, originally came to the clinic for examination in April 1948, her complaint being pain in the chest and shoulder. At that time, she had no symptoms referable to the gastrointestinal tract other than an occasional gaseous distention of the abdomen. No family history of colon disease was obtained, and the rectal examination was negative. Her next visit was in October 1948. On this occasion, she reported a mild diarrhea and a sensation of discomfort in the rectal area. She was advised to return for a stool test and colon roentgenogram, but failed to do so.

The patient was not seen again until August 1953, when she was visiting her sister in the hospital following the latter's colectomy. In view of the strong family history of polyposis reported by the sister (chart I), she decided to have an examination. Several small rectal polyps were discovered on proctoscopy. The roentgenogram revealed a polyposis of the entire colon (fig. 3). The rectal polyps were fulgurated at that time.

* From the Sanders Clinic and the Baptist Memorial Hospital, Memphis, Tennessee.

CHART I

Ancestry of Cases I, II, III, IV, & V



On Oct. 26, 1953, a subtotal colectomy and ileosigmoidostomy were performed, an end to end anastomosis being made 12 to 14 cm. from the dentate line. The patient was allowed to return home 2 weeks later.

The pathologist reported multiple polyposis without evidence of carcinoma (fig. 4).

At her next observation, on November 27, 1953, 1 small rectal polyp was fulgurated. Since she lived in a distant city, she was referred to her home physician for a close follow-up, including a proctoscopic examination every few months. Her sister reported that she was well and free of disease 2 years postoperatively.

Case 3. W. R., a 42 year old white woman, came to the clinic in 1947, complaining of rectal bleeding and a cramping pain in the lower abdomen and across the back. Eighteen months earlier she had had 57 rectal polyps fulgurated elsewhere. Thereafter, she had remained free of symptoms until 2½ months previously, when the rectal bleeding began. Her physician had made a colon roentgenogram, which demonstrated polyps beyond the reach of the proctoscope. It was learned that several members of her family had had polyposis and cancer of the colon (chart I).

The only noteworthy finding on physical examination was a slight pallor.

Operation was performed on Feb. 27, 1947, the terminal ileum and colon being removed and an ileosigmoidostomy made. The patient's postoperative course was satisfactory.

On examination of the specimen, the pathologist found an extensive polyposis, with histologic malignancy but no evidence of invasion.

The patient returned 3 months later and several rectal polyps were fulgurated. At an-

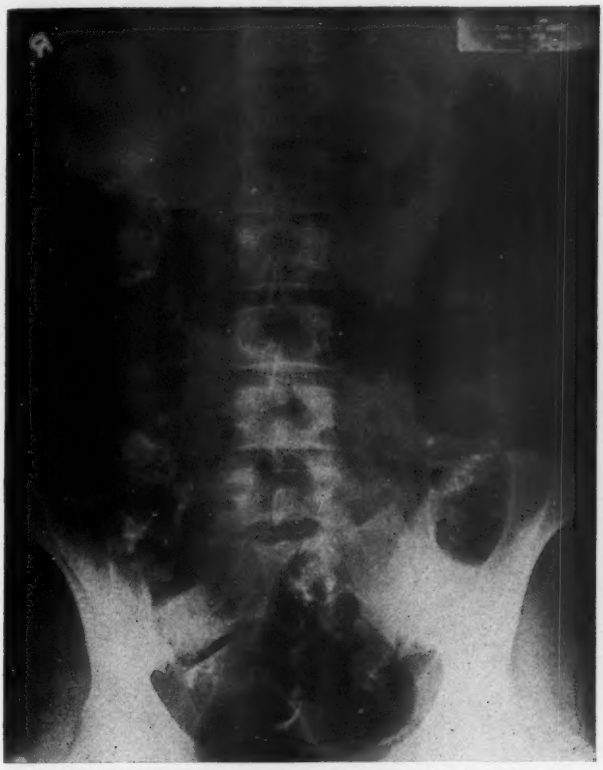


FIG. 1. Case 1. Contrast enema showing multiple polyposis

other examination on Dec. 13, 1948, roentgenograms made following a contrast enema exhibited no evidence of polyp formation. She had passed a little blood after the last fulguration a few days previously, but no explanation for this was found.

She was seen again on Oct. 2, 1951, and at this time a complete obstruction at the site of the anastomosis was demonstrated in the roentgenogram. At operation, the sigmoid was entirely encircled by a constricting growth. The terminal ileum and sigmoid were resected and the bowel reunited end to end.

The pathologist's diagnosis was adenocarcinoma extending through the bowel wall. None of the 10 resected nodes were involved.

On her last visit, Nov. 18, 1952, the patient complained of severe back pain. A complete examination failed to elicit any sign of recurrent or metastatic malignancy. We have been unable to obtain any recent information regarding her condition.

Case 4. P. C., a white woman, 36 years of age, came for examination on Feb. 6, 1947, because of rectal bleeding. She gave a history of intermittent attacks of rectal bleeding and intestinal cramping since the age of 11 years, and of having been treated at intervals for amebiasis. A proctoscopic examination had been made for the first time in 1946, and on that occasion multiple polyps were found in the rectum and lower sigmoid. A roentgen study had revealed a marked polyposis extending to the cecum. The patient also gave a strong family history of familial polyposis and colon disease (chart I).

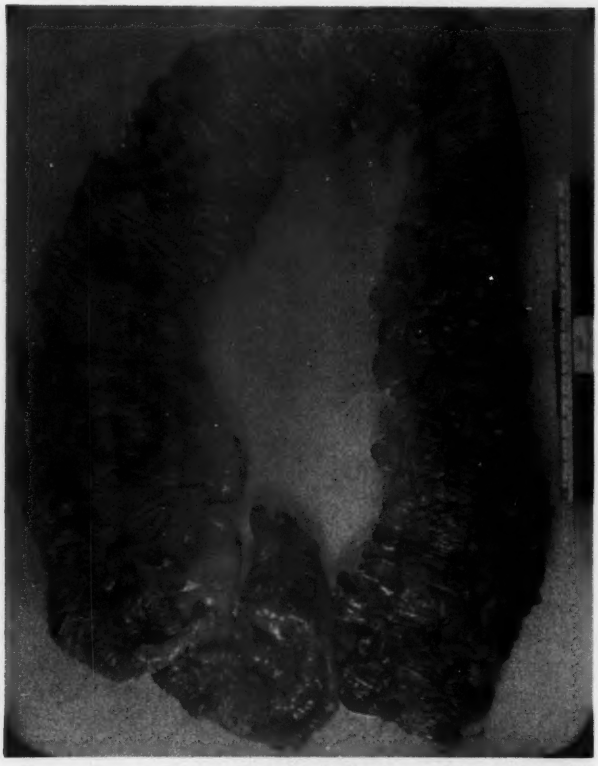


FIG. 2. Case 1. Resected specimen showing multiple polyposis

The physical examination was negative. On proctoscopy, the rectum and rectosigmoid were literally covered with polyps.

On Feb. 13, 1947, an ileostomy with a skin graft covering was made. After leaving the hospital, the patient vomited frequently for 4 weeks and had to be readmitted. The vomiting finally subsided under treatment by nasogastric suction.

On July 8, 1947, she returned with a definite obstruction of the small intestine. Ten inches of the terminal ileum were removed in releasing the obstruction. The appearance of the colon was good, although the multiple polyps found at the first operation could still be palpated. Nothing further was done at this time, the patient being advised to have the colon resected later.

At her next visit, Nov. 8, 1947, she complained that the ileostomy did not function well and it was necessary to resort to enemas for elimination. She was most anxious to have the continuity of the bowel restored, stating that she was willing to accept a shorter life with the fear of malignancy in order to be rid of the ileostomy.

An ileosigmoidostomy was performed on Nov. 19, 1947. At operation, 1 large, firm polyp, apparently malignant, was found in the transverse colon. A resection was planned for 3 weeks later.

Following her readmission to the hospital, Dec. 19, 1947, the terminal ileum was excised and a subtotal colectomy performed. Pathologic study revealed an adenocarcinoma originating in a glandular polyp, and multiple polyps which were histologically malignant.

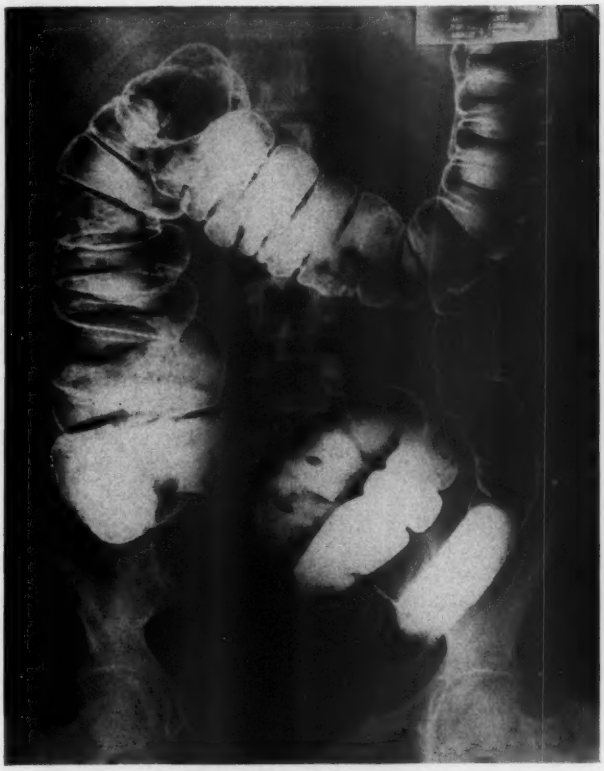


FIG. 3. Case 2. Contrast enema showing multiple polyposis

The patient was seen again on March 4, 1949, her complaint then being edema of both legs and ankles, of 3 weeks' duration. The physical examination with respect to the colon disease was negative. One year later she returned because of abdominal pain, nausea, and vomiting. She had had no diarrhea or bleeding. No specific diagnosis was made.

Not until Dec. 17, 1953, was the patient seen again. At that time she still complained of tenderness across the lower abdomen, and reported that she had been passing red blood in the stools for 6 months. A complete physical examination revealed nothing of significance other than a pallor of the mucous membrane. Proctoscopy of the rectum and rectosigmoid was negative, and no evidence of malignancy was seen in roentgenograms of the gastrointestinal tract and lumbar spine. The patient was advised to enter a hospital for treatment of a drug addiction, although this advice was refused. She is still under observation having been seen Feb. 19, 1954, at which time there was no evidence of malignancy.

Case 5. T. McC., a 22 year old white man, was first observed on Dec. 12, 1948, his complaint being an abdominal mass of several months' duration. He also had a moderate diarrhea, with the passage of 4 to 5 stools daily and small amounts of mucus, but no blood. According to his history, his symptoms began with the onset of diarrhea at the age of 15; later, the diarrhea was accompanied occasionally by the passage of blood. In 1945, during his service in the Navy, a diagnosis of familial polyposis was made and a colectomy with ileosigmoidostomy was performed. In addition, polyps in the rectum and lower sigmoid

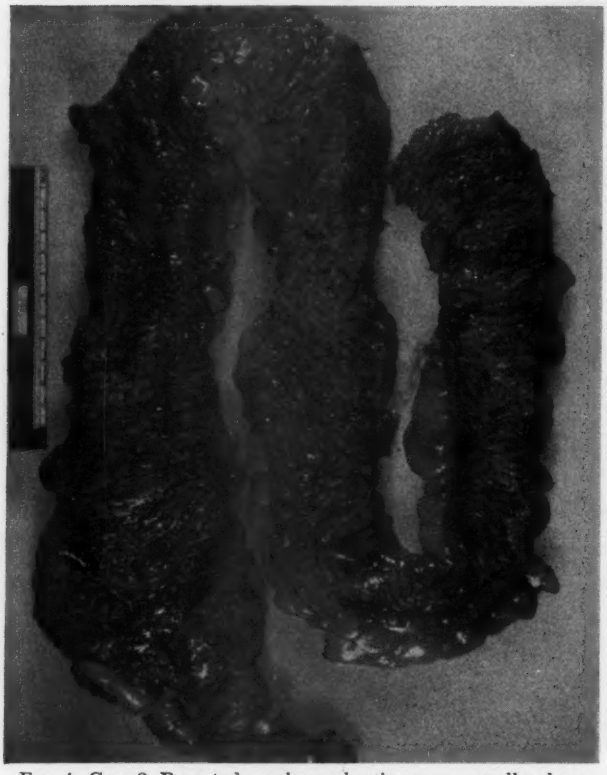


FIG. 4. Case 2. Resected specimen showing many small polyps

were fulgurated. He recovered satisfactorily and subsequently was discharged from the Navy.

In 1947, he developed an acute small bowel obstruction which necessitated surgical release. No mass was found at that time.

In February 1948, he discovered an abdominal mass and was explored at another hospital. The mass proved to be an inoperable growth in the colon. Following his recovery from the operation, he received 20 roentgen treatments over the area of the mass.

On his visit to the clinic, he reported a strong family history of polyposis and cancer of the colon (chart I).

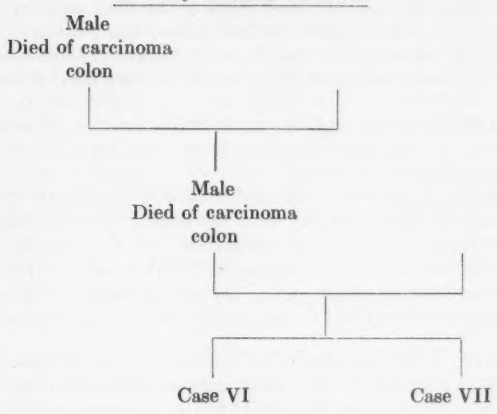
Physical examination revealed evidence of a slight weight loss. There were 4 scars in the abdominal wall, and just to the right of the midline a large, slightly movable mass was palpated. On proctoscopy, several small polyps were found in the rectum, although none were seen in the rectosigmoid.

Further surgery was not advised, and the patient returned home. He died, apparently of carcinoma, in 1949.

Case 6. L. S., a white woman, 29 years of age, was admitted to the Baptist Hospital on Nov. 1, 1948, with a complaint of intermittent fecal incontinence for a period of 6 years. Her family history revealed that her father and grandfather had died of colonic cancer and a sister had polyposis (chart II).

CHART II

Ancestry of Cases VI & VII



On physical examination, the perineal body was badly torn and a marked rectocele was present. There were also several hemorrhoids, and on sigmoidoscopic investigation, numerous colon and rectal polyps were found. Roentgenograms demonstrated a polyposis of the entire colon.

The rectocele was repaired, and on dismissal from the hospital, the patient was advised to return within a few weeks for operation upon the colon. She was not seen again until April 12, 1951. At that time she still complained of fecal incontinence. The perineum was in good condition; otherwise, the physical findings were essentially the same as upon her first examination. The polyposis of the colon was again demonstrated by proctoscopic and roentgen studies.

On April 28, 1951, an ileostomy was made, the colon was resected to the sigmoid, and the distal segment was closed. The pathologist reported that the specimen contained many small polyps, 3 of the largest exhibiting early carcinoma.

The abdomen was reopened on Aug. 13, 1951, and a subtotal hysterectomy, bilateral salpingo-oophorectomy and abdomino-perineal resection of the remaining colon and the rectum performed. The pathologic diagnosis was polyposis and adenocarcinoma of the rectum with metastases to the mesenteric glands.

The patient recovered from the operation and did well for a while, but died of carcinoma approximately 1 year following the last operation.

Case 7. H. D., a 23 year old white woman, was first seen at the Baptist Hospital on Oct. 28, 1952. She complained chiefly of diarrhea and tenesmus of 3 years' duration, but also stated that she had had 3 or 4 episodes of rectal bleeding during the previous 3 months, and within the previous month had lost 20 pounds in weight. It was learned that her father and sister had trouble of a similar nature, and her paternal grandfather had died of cancer of the colon (chart II).

The physical examination was negative. Proctoscopic study disclosed a large ulcerated mass in the anterior rectal wall and numerous polyps throughout the rectum. A diagnosis of carcinoma was made from a biopsy of the ulcerated mass.

A total rectocolectomy with ileostomy was performed on Nov. 27, 1952. A solitary metastasis was excised from the liver. In addition, it was necessary to perform a panhysterectomy and partial vaginectomy because of extension of the rectal lesion.

On examination of the colon, the pathologist found multiple polyps involving the entire colon, as well as the malignant polyp of the rectum.

The patient was dismissed from the hospital 3 weeks postoperatively, apparently in good condition, but died 18 days later. The cause of her death is not known.

Case 8. E. W., white man, aged 39 years, came to the clinic on March 23, 1949, because of rectal bleeding of 10 years' duration. In 1942, he had had a hemorrhoidectomy for this complaint. In 1944, he was seen by other physicians, who had made a diagnosis of polyps of the colon and fulgurated several of the growths. He reported that his brother also had polyposis.

There were no significant findings on physical examination. Proctoscopic inspection revealed multiple polyps, and roentgenograms made following an air contrast enema demonstrated polyposis of the entire colon.

Polyps were fulgurated on several occasions from 1948 to 1951. On Sept. 5, 1951, a subtotal colectomy and ileosigmoidostomy were performed. The pathologist reported that the specimen contained numerous polyps throughout, but no evidence of carcinoma.

Other polyps were fulgurated on 3 occasions from 1951 to July 16, 1954; on the latter date the distal segment was free of disease. In a letter dated March 4, 1955, the patient stated that he felt well but was still having 3 bowel movements daily and was troubled with gaseous indigestion.

Case 9. J. W., a 47 year old white man, was first seen on June 16, 1954. For approximately 1 year he had had 10 to 12 stools daily, although without melena. He also complained of moderate belching and distention. His family history revealed that a brother (Case #8) had polyposis and a sister had chronic ulcerative colitis.

Aside from several small rectal polyps which could be felt with the finger, the physical examination was negative. On proctoscopy, multiple polyps were found in the rectum, and polyps were seen throughout the colon in the roentgenogram.

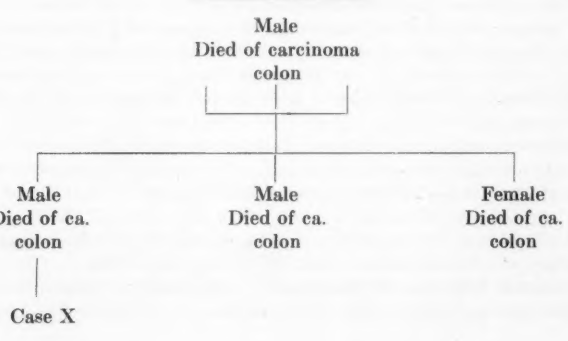
A colectomy was advised, but the patient preferred to have the operation at another hospital. His brother informed us that a subtotal colectomy with ileostomy was performed in August 1954, and in addition to multiple polyps, the colon contained a carcinoma at the splenic flexure. A septicemia, together with cerebrovascular accident, was responsible for his death approximately 1 month following the operation.

Case 10. E. E. D., a 25 year old white woman, came to the clinic on April 13, 1953, complaining of abdominal pain of 2 years' duration. The pain was located chiefly in the right upper quadrant, was episodic and colic-like in character, and was aggravated by greasy foods and fats. In addition, during the previous 2 years she had been troubled with a rather severe diarrhea, frequently having 6 to 8 stools daily. Her grandmother, father, an aunt, and an uncle had died of carcinoma of the colon with polyposis (chart III).

Multiple rectal polyps were observed through the proctoscope, but none could be seen

CHART III

Ancestry of Case X



above the rectum in the air contrast roentgenogram. A nonfunctioning gallbladder was shown in the cholecystogram.

A cholecystectomy was performed on April 22, 1953, and at the same time the colon was explored. No polyps could be palpated, although a mass was felt in the rectosigmoid area. Three weeks later, after preparation of the colon, an anterior resection of the sigmoid was performed. A subtotal hysterectomy and bilateral salpingo-oophorectomy were also necessary because of an extensive pelvic endometriosis. The colon mass proved to be an endometrioma, however, the pathologist found multiple polyps in the mucosa of the resected specimen.

The rectal polyps were fulgurated following the patient's recovery. A total colectomy with ileorectostomy was carried out in January 1956 with an uneventful recovery.

The pathologist reported multiple polyps in the colon with no evidence of carcinoma.

DISCUSSION

Peutz[13] described a syndrome of melanosis of the oral mucous membranes with polyposis of the intestines, and Jeghers, and associates[9] observed melanosis of both the oral mucosa and digits in association with intestinal polyposis. None of our patients had polyps in regions other than the colon, and none had pigmented areas in the buccal mucous membranes or digits. Nor did any of the group have multiple sebaceous cysts in combination with colonic polyps, as recently described by Oldfield.[12]

It should be emphasized that multiple polyps and pseudopolyps, found after long standing inflammation of the colon, are similar in some respects to familial polyposis, but are, in reality, a different disease. In both conditions, however, there appears to be a propensity to malignant degeneration.

An occasional patient with polyposis will meet disaster from hemorrhage, perforation, or intussusception; the principal danger, however, is the development of a carcinoma. The exact percentage of patients who develop carcinoma is not known, since the number followed without treatment has been too few and the period of their observation too short to permit an accurate estimate. Many observers are of the opinion that 100 per cent of patients with familial polyposis will eventually have carcinoma if untreated, although this has not been borne out by case studies. McKenny[11] followed the histories of 3 families over a long period and found that 38 per cent of those who had polyposis developed cancer. In 1907, Doering[4] reviewed 50 cases of polyposis; the cause of death was known in 36 of these patients, and of these, 21 died of cancer. Hullsiek[8], in 1928, found carcinoma in 36 per cent of 127 patients. Invasive carcinoma was either present at the time of treatment or developed subsequently in 7 of our 10 patients.

Smith and Hill[16] postulate that just as certain families have an inherent trait toward polyposis some may also have a trait toward malignancy in the polyps. They base this upon the fact that cancer developed frequently in some of the families they observed, while in another family there were several individuals beyond the age of 50 who had polyposis but no evidence of carcinoma.

Thus, it appears that the danger of carcinoma probably is not 100 per cent, yet is sufficiently serious to warrant radical surgery in almost every case.

Diagnosis: The symptoms of familial polyposis vary in different patients.

Some have only mild, if any symptoms, whereas others have severe, bloody diarrhea, malnutrition, abdominal pain, and tenesmus. Occasionally, the first indications of the disease arise from a carcinoma superimposed upon the polyposis; these may consist of pain and distention from colon obstruction.

In uncomplicated cases, physical examination reveals only palpable polyps in the rectum, or, the growths may be so small or so few in number as to be undetected digitally.

Laboratory studies are not specific. Growths in the lower sigmoid and rectum may be visualized through the sigmoidoscope, and colon roentgenograms, particularly of the air contrast type, will usually show numerous polyps of various sizes (figs. 1 and 3).

Treatment: While it is recognized that polyposis calls for surgical intervention, there is some disagreement as to the choice of procedures. The advantage of subtotal colectomy with ileosigmoidostomy or ileoproctostomy lies in the fact that sphincteric control is retained, the number of stools is low, and little or no nutritional problem arises following operation. In addition, the remaining segment of the colon can be observed through the sigmoidoscope, permitting fulguration of residual or subsequent polyps. By this procedure, however, potentially malignant tissue is left in the body and many patients will not cooperate by having proper follow-up proctoscopic examinations.

Hoxworth and Salughter[7] reviewed the cases of 35 patients on whom surgery had been performed. Seventeen of the patients had had ileosigmoidostomy with colectomy. At that time, 4 of the 17 had died of carcinoma which developed in the remaining segment from 1 to 6 years after fulguration was begun.

Jones[10] reported the case of a patient in whom the lower segment was fulgurated for 2 years; thereafter a colectomy and ileosigmoidostomy were done. The patient died of carcinoma 2 years later. A similar experience led Ravitch and Sabiston[14] to advocate anal ileostomy and total rectocolectomy. Cattell[3], who also had such a case, advocates abdominal ileostomy with total rectocolectomy. Schutte[15] reported 2 cases of patients in whom carcinoma developed in the residual rectum, and 2 of our 10 patients had a cancer in the stump of sigmoid following ileosigmoidostomy.

Smith and Hill[16], on the other hand, report that of 25 patients who had colectomy and ileosigmoidostomy at the Mayo Clinic, only 2 subsequently died of cancer. One of the 2 had an invasive cancer at the time of the original operation. The other later had a cancer of the residual sigmoid in an area which could not be reached by the sigmoidoscope. Two other patients died of unrelated causes. Thus, 21 were alive and apparently free of the disease after having been under observation from 6 to 19 years.

Total rectocolectomy with an abdominal ileostomy in the treatment of polyposis possesses the advantage of total extirpation of all colon tissue, which obviates the possibility of carcinoma. It has the disadvantage of being more formidable surgery than subtotal colectomy, and a higher mortality rate can be expected. Further, despite careful management, many patients with an ileostomy have trouble with excoriation of the skin and obstruction of the stoma, as well

as the difficulty of living with an ileostomy. Malnutrition, also, is often a problem.

Anal ileostomy, recommended by Ravitch, does not seem to be the answer, as evidenced by the report of Carlson and Novacovich[2]. These authors performed colectomy with anal ileostomy in 3 patients who had polyposis. Two of the patients had a long and difficult postoperative course, and 18 months after the final operation still had involuntary stools at night. The third patient later had to have the anal ileostomy converted into an abdominal ileostomy. They mentioned a fourth case which was also a failure. Goligher[6] likewise reported poor results in 2 cases following the Ravitch type of operation.

The controversy regarding the most desirable operative procedure for polyposis of the colon will not be settled until more patients have been treated and a sufficient time has elapsed to permit an evaluation of the various methods employed. At present, our preference is subtotal colectomy with an end to end anastomosis of the ileum to the rectum as low as is technically possible. If the patient's condition permits, the operation is completed in one stage. We believe that, in the majority of patients, low resection and anastomosis, with a close follow-up and fulguration, will give adequate protection against carcinoma and will afford the patient satisfactory living conditions. Those patients wherein the distal rectum contains an invasive malignancy or a sheet of polyps which makes fulguration impossible or impracticable are exceptions to this method of treatment. Another exception is the patient, who, because of a psychopathic personality, lack of education, or some other reason, cannot be expected to return for the necessary observation.

A procedure which we have not had occasion to attempt, but which seems promising, has been described by Aylett[1]. In 1 patient, he anastomosed the ileum to the distal rectum by a combined abdomino-perineal approach. Only about 1 centimeter of rectal mucosa was left intact, yet the patient subsequently had complete continence.

SUMMARY

Familial polyposis of the colon is inherited as a Mendelian dominant or recessive. It is transmitted by both sexes, and usually appears at puberty or in the second or third decades of life.

Carcinoma develops in a high percentage of patients constituting a threat to life.

At present, the choice of surgical treatment lies between subtotal colectomy with ileoproctostomy and fulguration of polyps in the distal segment, and total colectomy with abdominal ileostomy.

899 Madison Ave.
Memphis 3, Tenn.

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KARTAGENER'S SYNDROME

REPORT OF A CASE TREATED BY MIDDLE LOBE LOBECTOMY

THOMAS C. MOORE, M.D., AND RICHARD A. SILVER, M.D.*

Muncie, Ind.

The frequent association of situs inversus, bronchiectasis and sinusitis was first emphasized by Kartagener[6] in 1933. He reported 11 instances in 3 reports between 1933 and 1935[6, 7, 8]. His reports and subsequent observations have been regarded as strong support for those theories suggesting a congenital etiology for bronchiectasis.

Up to the present time, 105 cases of Kartagener's Syndrome have been reported[15]. Three reports in which this association occurred appeared in the literature prior to Kartagener's first publication; by Siewart[14] in 1904, Oeri[12] in 1909 and Guenther[4] in 1923. The largest series, of 14 cases each, have been recorded by Olsen[13] and by Torgersen[16, 17].

The findings of Adams and Churchill[1] and of Olsen are of considerable interest. Adams and Churchill, in 1937, found that 5 of 23 patients with situs inversus, studied at the Massachusetts General Hospital, also had sinusitis and bronchiectasis (21.7 per cent). This unusually high incidence was compared with the occurrence of sinusitis and bronchiectasis in the total number of patients registered during this period. This was found to be only 0.3 per cent. Similar findings were reported by Olsen in 1943. He found that 85 cases of situs inversus had been studied at the Mayo Clinic during a period of 22 years. Fourteen of these patients (16.5 per cent) also had bronchiectasis and sinusitus. During this period only 0.5 per cent of the patients registered at the Mayo Clinic were found to have bronchiectasis.

Multiple instances in one family of situs inversus and of Kartagener's triad have been found. Notable are the reports of Lopez Areal[10] and of Torgersen[16]. Lopez Areal reported in 1944 that 3 of 11 children in one family had situs inversus and bronchiectasis. The parents were first cousins. Torgersen encountered situs inversus in 3 of 5 children in one Norwegian family.

Pulmonary resection was carried out in only 9 of the patients of the 105 reported cases[1, 2, 3, 5, 9, 11, 15]. A number of the cases were recorded as necropsy finding. Some were studied prior to current acceptance of resection for bronchiectasis. Others were reported as too advanced to benefit from resection or too early or minimal to justify this procedure. Due to the rarity of this triad and the limited number of reported cases in which the patient was managed by pulmonary resection, it has seemed desirable to report a case of Kartagener's triad with middle lobe bronchiectasis in a 20 year old man treated by middle lobe lobectomy.

* From the Surgical and Radiological Services, Ball Memorial Hospital, Muncie, Indiana and the Department of Surgery, Indiana University School of Medicine, Indianapolis, Indiana.

CASE REPORT

The patient, a 20 year old white man, was admitted to the Ball Memorial Hospital on June 24, 1955 because of recurrent upper respiratory infections and roentgenographic evidence of situs inversus and middle lobe infiltration. He had had sinusitis and almost continuous respiratory infections for as long as he could remember. He had been confined to bed on several occasions with pneumonia. He coughed frequently and the cough was productive of a mucopurulent type of sputum. There was no history of hemoptysis. There were no significant findings on the routine physical examination. Laboratory examination of his blood revealed a hemoglobin of 16 Gm. per 100 cc., a red blood count of 5.0 million per cu. mm. and a white blood count of 9,600 per cu. mm. Twenty-two per cent of eosinophils were found on differential study of the blood smear.

Roentgen study of the patient's sinuses revealed normal frontal sinuses. Both antra showed membranous thickening which was more marked on the left side. The ethmoid sinuses were well developed and cloudy.

Bronchoscopy was carried out on June 25 under topical anesthesia with cocaine. There was no evidence of inflammatory or neoplastic obstruction of any of the bronchi examined. A moderate amount of mucopurulent material was aspirated from the area of the middle lobe bronchus on the left side. A radiopaque rubber catheter was left in the trachea on withdrawal of the bronchoscope. Bronchograms were obtained by injection of 10 cc. of Iodochlorol through this catheter after it has been passed fluoroscopically into the main bronchus on the left side. Bronchiectatic dilatation of the bronchi to both the medial and lateral segments of the middle lobe was shown (fig. 1).

The patient was taken to the operating room on June 27 and the left chest was entered through the bed of the resected sixth rib. Marked adhesions between the lung and the chest wall were sharply limited to the area of the middle lobe. The upper and lower lobes were free of adhesions. A good fissure was encountered between the middle and lower lobes. The fissure between the middle and upper lobes was incomplete. The middle lobe was removed

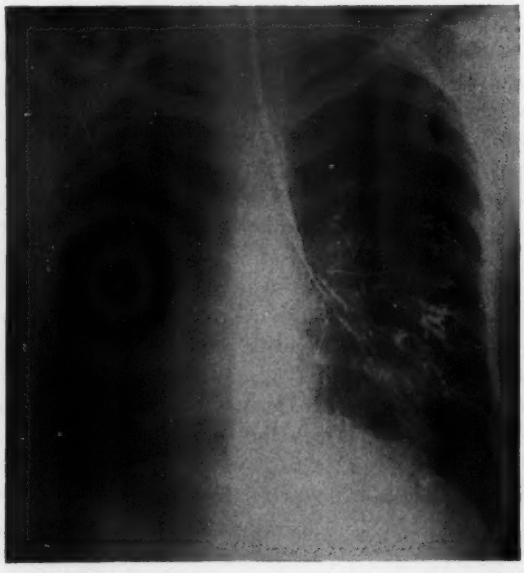

FIG. 1. Bronchogram showing bronchiectatic middle lobe (arrow) in the left hemithorax

in the usual manner by careful dissection, ligation and division of the major arteries and veins supplying the lobe. The bronchus was transected and the stump was closed with interrupted sutures of no. 000 silk. The bronchus and artery to this middle lobe were quite normal. Venous drainage of the lobe was largely through the middle lobe branch of the superior pulmonary vein. There was one small vein which emptied into that segment of the superior pulmonary vein which drained the inferior portions of the upper lobe. The operation was tolerated well and the patient was returned to the ward in good condition. Chest suction was maintained for 48 hours following operation. The remaining lobes on the left expanded well. The postoperative course was uneventful and he was discharged from the hospital on July 4, seven days following operation.

The resected middle lobe weighed 82 grams and there was gross evidence of marked atelectasis and congestion. Microscopically, there was considerable pleuritis, pneumonitis, fibrosis, atelectasis and bronchiectasis.

SUMMARY

A case of Kartagener's triad of bronchiectasis, situs inversus and sinusitis in a 20 year old white man is reported. The bronchiectasis involved the middle lobe in the left hemithorax. The patient had suffered from a severe cough and almost continuous repiratory infections. Middle lobe resection was carried out successfully with improvement of the patient on the basis of short term observation. In only 1 other patient, the case reported by Adams and Churchill, has middle lobe lobectomy been performed in a case of Kartagener's Syndrome.

110 North Cherry Street
Munice, Indiana

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AN UNUSUAL COMPLICATION OF INTESTINAL INTUBATION

CASE REPORT†

ROBERT C. OVERTON, M.D., MILTON M. SELF, M.D., VAN KADEN, M.D., ARTHUR C. BEALL, JR., M.D.

Houston, Texas

Intestinal tubes are employed so frequently and with such effectiveness for intestinal decompression that their potential hazard often is disregarded. Recently we have encountered an unusual complication resulting from the use of a Harris tube, and the resulting tissue trauma was so dramatically demonstrated at surgery that it prompted this report.

CASE REPORT

M. T., a 60 year old white woman, was admitted to the Jefferson Davis Hospital, Houston, Texas, on April 22, 1955, because of obstipation and cramping abdominal pain. The patient had suffered from constipation for many years and on several occasions had experienced abdominal pain and occasionally nausea and vomiting. The present illness began 3 days prior to admission with a similar sequence of events. The past history was significant in that appendectomy and uterine suspension had been performed 26 years previously.

Physical examination revealed a well developed, poorly nourished white woman in no apparent distress. The blood pressure was 116/70, pulse 88 per minute, and temperature 98.6 F. There was slight abdominal distention and a well-healed midline low abdominal scar and hyperperistalsis. The abdomen was not tender and no masses were palpable.

Laboratory studies were as follows: erythrocytes 4,200,000 per cu. mm., hemoglobin 13 Gm. per 100 cc., hematocrit 40, leukocytes 8,000 per cu. mm. with a normal differential count. Stool examination for occult blood was negative. Proctoscopic examination was negative to 20 centimeters. A diagnosis of mechanical small intestinal obstruction was made and a Harris tube was inserted. The patient improved rapidly with subsidence of distention and abdominal pain. Roentgenographic examination of the abdomen demonstrated only slight dilatation of the small bowel, the Harris tube having progressed to approximately midileum. Suction was discontinued and the tube clamped. Twenty-four hours later oral liquids were well tolerated. An attempt was made to withdraw the intestinal tube 72 hours after its introduction, but despite moderate traction it could not be removed. To maintain a more prolonged pressure the tube was taped to the nose with gentle traction, again without success. A 12 ounce weight was attached to the proximal end of the tube for 24 hours, but withdrawal was not effected, so the tube then was cut at the nose to allow its passage by rectum. Twenty-four hours later the tube had not moved, as indicated by a roentgenogram, and there were recurrent manifestations of intestinal obstruction with probable peritonitis.

At operation on April 27, 1955, there was generalized peritonitis and complete obstruction in the midileum due to an adhesive band. The source of the peritonitis was found to be a perforation of the mesenteric aspect of the ileum proximal to the obstructing point. Closer inspection revealed ulcerations along both mesenteric and antimesenteric borders of the intestine proximal to the point of obstruction. A majority of the erosions were mucosal in depth but many extended through almost the entire thickness of the wall. The linear configuration of the ulcers at fixed points suggested the intestinal tube as the source of

† From the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine, and the Surgical and Pathological Services of the Jefferson Davis Hospital, Houston, Texas.

ulceration. Indeed, in several areas an actual imprint of the tube was formed by narrow ulcerations of 10 to 15 centimeters in length (fig. 1). The distal end of the tube was fixed at an angulation in the bowel immediately proximal to the obstructing band, and its removal was accomplished only by division of the bowel during resection of the perforated segment.

FIG. 1. Representative section of small intestine demonstrating linear ulcerations produced by pressure necrosis.

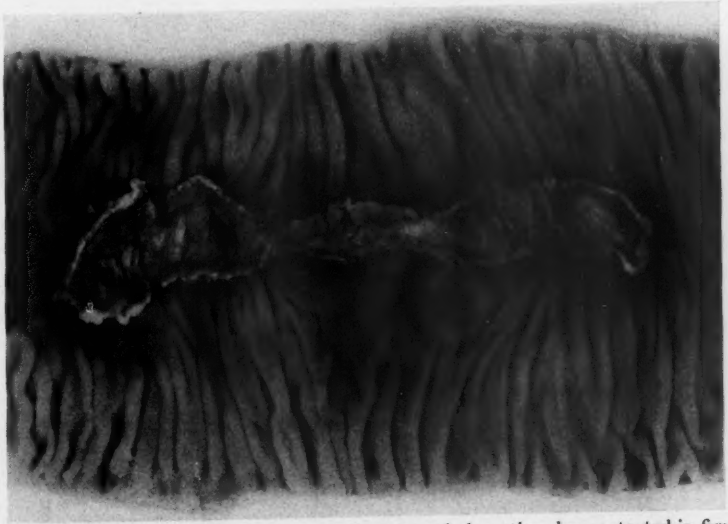

FIG. 2. A higher magnification of one of the areas of ulceration demonstrated in figure 1. Bowel wall perforation occurred in a similar area.

Approximately 3 feet of the bowel containing the perforated area and the majority of the deeper ulcerations was resected and an end to end anastomosis performed. Postoperatively the patient improved rapidly and was discharged on the eighth postoperative day.

The resected segment of small intestine was 75 centimeters in length. Scattered along the mesenteric border and in a few areas between the mesenteric and antimesenteric borders were ovoid areas of yellow-green discoloration of the serosa. These areas corresponded to longitudinal ovoid mucosal defects varying from 11 by 1.0 cm. to 1.0 by 0.6 cm. and covered with a green or yellow, ragged, friable membrane. The intervening mucosa was edematous with foci of hemorrhage in the submucosa adjacent to the areas of ulceration (fig. 2).

Microscopically, there were rather sharply defined areas of ulceration of the mucosa with dense infiltrations of neutrophilic granulocytes, and less numerous lymphocytes and macrophages enmeshed in fibrin and cellular debris. In some areas this process extended throughout the entire wall. In adjacent uninvolved portions there was extensive hyperemia and recent hemorrhage in the submucosa.

DISCUSSION

This unfortunate complication of intestinal intubation is rare, and reflects misuse of the tube rather than a fault of the appliance. It is apparent that the marked trauma to the bowel in this case resulted from fixation of the tube proximally with the distal end already fixed. The resulting erosion is readily understood. Plication of 18 or 20 feet of bowel on a taut 3 foot hard surface tube was obviously inviting the resultant tissue abuse. Nasal, pharyngeal, and esophageal erosions are frequent from the hard surface of tubes in the absence of taut fixation[10].

It is unusual to experience difficulty in the removal of a tube of this type. The majority of complications in which removal has been difficult or impossible has been associated with the balloon attached to the distal end. Such complications occur with use of Cantor, or Miller-Abbott type tubes when over-distention of the balloon from permeation of the wall with intestinal gases occurs, or when a portion of the balloon engages in an anatomic *"bottle neck"* such as the iliocecal valve[4, 13, 11, 15]. In this case a Harris tube, a short, single lumen tube without an attached balloon, was employed, and its freakish engagement at an angulated point in the bowel is most unusual.

The literature contains few references to actual perforation of a viscus by an intestinal tube. Eliason and Welty[5] report a case of perforation of the esophagus and Holinger and Loeb[8] a similar occurrence, although it is difficult to exclude spontaneous rupture of the esophagus from persistent vomiting in these patients. Mahon[12] and Cheffe[3] each observed cases of a perforated stomach by an intestinal tube, but mention was made of neoplastic involvement of the stomach at the site of perforation. In none of the above described cases was the perforation associated with difficulty in the removal of the tube. Berger and Achs[1], however, report a case in which perforation of the small bowel was demonstrated at laparotomy necessitated by the inability to remove or pass per rectum a Miller-Abbott tube. Kaplan and Michel[9] and Schlicke and associates[14] have reported cases of bowel rupture at sites of engagement of distended bags. Warren and Cattell[16] experienced a fatality when an intussusception produced by a tube perforated. That such complications occur more frequently than the literature

would suggest is supported by the comment of Harris[6] that he has received several personal reports of deaths resulting from necrosis and perforations of the bowel when bags attached to intestinal tubes had become distended and caught.

Prevention of this complication is easily accomplished by more meticulous and judicious use of these appliances. Cantor[2], Harris[7], and others have listed the dicta necessary to obviate such a complication in the majority of instances. If difficulties are encountered in the removal of an intestinal tube, traction or forceful pressure should never be applied, as this case so well demonstrates. If the tube cannot be removed easily it should be cut and allowed to pass by rectum, its course during this descent being carefully followed by repeated roentgenograms. Failure of the tube to pass or evidence of increasing obstruction dictate immediate laparotomy.

Baylor Univ.
College of Medicine
Houston, Texas

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EPITHELIOMA OF THE JEJUNUM

MILTON W. DURHAM, M.D., WALTER HART, M.D.

Spokane, Wash.

Carcinoma of the jejunum occurs infrequently; the reported incidence varies depending upon whether the cases are derived from clincial or autopsy series. Jejunal carcinoma was reported in 2.1 per cent of Hunt and Kaneb's series[4] of 147 operative cases of gastrointestinal carcinoma and in 0.15 per cent of all carcinoma of the gastrointestinal tract as reported by Mayo and Nettrour[6].

The most common malignant lesions of the jejunum are adenocarcinomas, similar to those occurring in the large bowel. Collected series have shown that adenocarcinomas predominate in the jejunum, a slightly lesser number comprises the sarcomas with the argentaffinomas occupying an intermediate position. However, epithelioma of the jejunum is an extreme rarity. One case has been reported by Mayo and Nettrour in a series of 40 malignant tumors of the jejunum and another, more recently, by Prat and Dominguez[7]. In view of this infrequency, we would like to add our case.

CASE REPORT

W. Y., a 34 year old white mail-carrier was first examined on April 10, 1950, complaining of dizzy spells, weakness and shortness of breath. His symptoms had developed gradually during the previous few weeks and at the time of examination he was so weak that it was difficult for him to do his work. He denied gastrointestinal symptoms. Physical examination revealed a slightly obese man who was very pale. Other than signs of anemia, the physical examination was negative. The hemoglobin was 7.5 Gm. per 100 cc. and the red blood cell count was 2,420,000 per cu. mm. On April 12, before diagnostic studies were completed, the patient developed severe abdominal cramping pain associated with projectile vomiting. A barium swallow showed complete obstruction of the small bowel at the duodenojejunal junction.

Laparotomy was performed for a high intestinal obstruction. Upon opening the peritoneum there was a small amount of free fluid. Immediately presenting within the abdomen was a massive intussusception. Approximately 3 feet of small bowel had progressed upwards to the ligament of Treitz as the intussusceptum could not travel downward because of its anatomic fixation at the duodenojejunal junction. After reduction by taxis, the cause of the intussusception was found to be a tumor located 8 inches below the ligament of Treitz. It appeared to extend completely through the muscular wall with dimpling of the serosa on the antimesenteric side. This portion of the jejunum was resected, the upper level of resection being at the duodenojejunal junction. A wide portion of mesentery was resected with the tumor and immediate examination of its nodes revealed none positive for metastases.

Figure 1 shows the serosal and mucosal aspects of the fungating gray lesion, which measured about 5.5 cm. in diameter. Microscopically, the tumor was composed of highly anaplastic epithelial cells of squamous type (fig. 2). They were characterized by marked variations in size and shape. Their nuclei demonstrated marked hyperchromasia and many cells possessed giant nuclei. In occasional areas intercellular bridges were demonstrable and many cells in the lesion appeared to be undergoing keratinization. Sections taken from the edge of the tumor failed to demonstrate a gradual transition from normal mucosa to

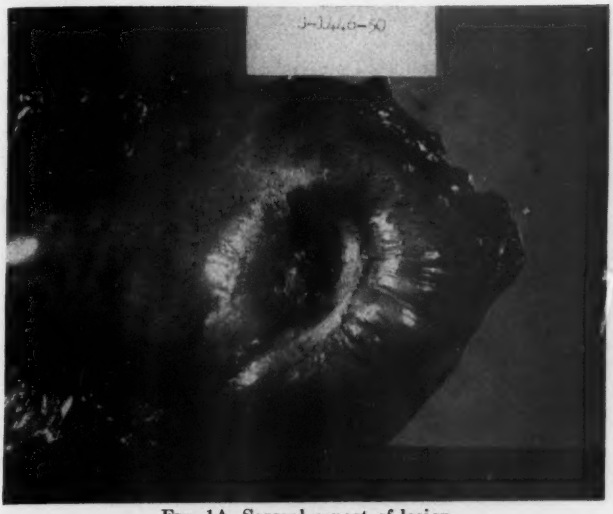

FIG. 1A. Serosal aspect of lesion

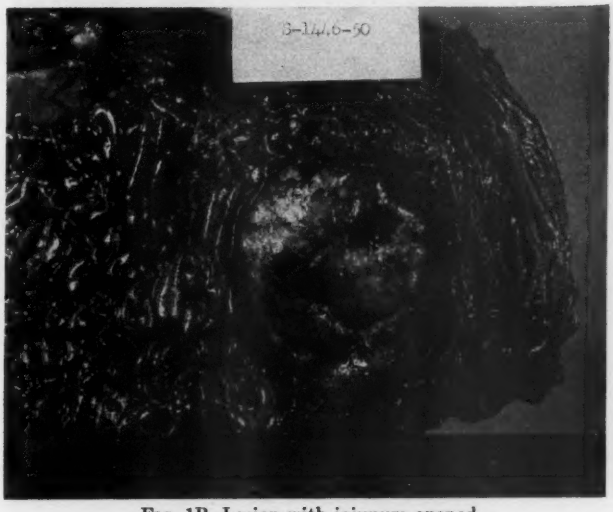

FIG. 1B. Lesion with jejunum opened

anaplastic tissue. Sections of regional lymph nodes showed no metastases. The diagnosis was squamous cell epithelioma (grade III) of the jejunum.

Postoperative convalescence was uneventful but within 1 month there developed recurrent cramping abdominal pain. On June 13, another exploration was done because of complete small bowel obstruction. There was no local recurrence of the tumor in the bowel but rather there were massively enlarged retroperitoneal lymph nodes which completely

FIG. 2A. Microscopic appearance under low power

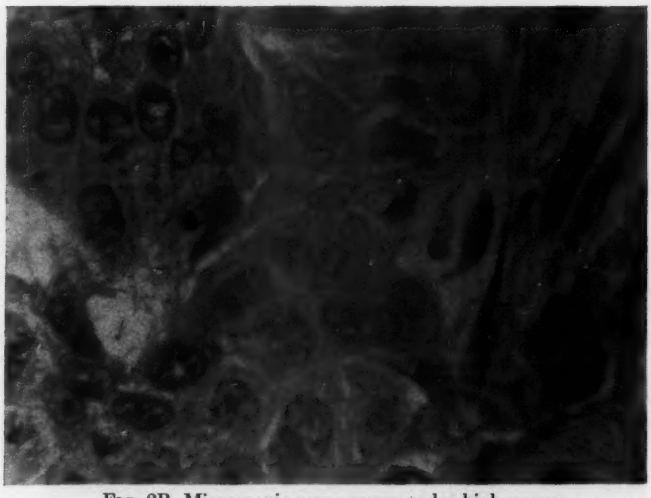

FIG. 2B. Microscopic appearance under high power

obstructed the upper jejunum by pressure. A by-pass was constructed. This temporarily relieved the symptoms of obstruction but the patient died Aug. 9, 1950.

An autopsy revealed massive retroperitoneal tumor masses identical microscopically to the original lesion. Careful examination of other sites for primary tumor was negative.

DISCUSSION

The infrequency of squamous epitheliomas of the jejunum applies also to the stomach, the rest of the intestinal tract and the biliary tree. A survey of 22,365

operations on the gallbladder and biliary tract at the Mayo Clinic from 1907 to 1930 revealed 212 cases due to neoplastic processes. Fifteen were a combination of squamous cell and adenocarcinoma but only 5 were squamous epitheliomas[3]. A case of squamous cell carcinoma of the pancreas was presented at the Massachusetts General Hospital in 1953. It was postulated that primary squamous cell carcinoma in this organ originated via squamous metaplasia, probably of the duct epithelium[1].

In a survey of epitheliomas of the stomach, O'Brien and Meehan found only 9 pure cases reported[6]. Diaz also has reported a similar case[2]. The latter, in his study of its etiology, talked with Duran-Jorda of England who believed that adenoacanthomas or carcinomas of stratified squamous epithelium originated from flat semisquamous layers which cover and protect all the mucous membranes of the body. This theory has not been widely accepted. Whether these tumors arise from intestinal epithelium via metaplasia or from congenital rests is certainly unsettled.

SUMMARY

A case of squamous cell epithelioma involving the upper jejunum is described. This rare tumor manifested itself by anemia and obstruction due to intussusception. Death occurred 4 months after resection and was due to retroperitoneal metastases.

820 South McClellan St.
Spokane, Washington

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EDITORIAL

MASSIVE UPPER GASTROINTESTINAL BLEEDING

Acute gastrointestinal hemorrhage is second to an acute condition within the abdomen as a cause of concern to the surgeon and internist. The main causes for severe upper gastrointestinal bleeding are esophageal varices, gastric carcinoma and duodenal ulcer. The latter is by far the most frequent of these. This discussion will be limited to the diagnosis and methods of treatment of these conditions. There are three paramount problems, namely: (1) the determination of the cause of the bleeding, (2) a decision as to whether conservative replacement of blood or operative measures are indicated, and if the latter course is to be followed, when is the optimum time, and (3) what safe procedures are available for the control of the bleeding and when should definitive therapy be instituted.

Some appreciation can be had as to the magnitude of the problem by realization that 25 per cent of the patients admitted to the hospital with ulcer are for varying degrees of bleeding. The majority are controlled without too much difficulty and many will stop without treatment. It is difficult to determine which will continue to bleed and may be impossible until after the ideal time for intervention has passed.

When a patient is seen with melena or hematemesis, of an unknown origin, it is incumbent on the attendants to determine the cause of the bleeding as soon as safety permits. The first concern in the presence of massive bleeding is restoration of the lost blood. An emergency gastrointestinal roentgenographic study should be done using great caution, keeping in mind that this is done with some risk. Unfortunately it is not always possible to obtain a completely satisfactory examination and the exact bleeding site will not be located in approximately 20 per cent of the patients.

Conservative medical management may result in a serious consequence in many severe bleeding ulcers. The opposite is equally true in patients who have conditions such as esophageal varices, hiatal hernia, blood dyscrasia and other systemic diseases, when early operation may be unnecessary or disastrous. A positive diagnosis is essential if proper treatment is to be rendered.

The age factor has been emphasized and overly so in some instances. It usually is considered that in the older patient the bleeding will not cease. Generally this may be true but more and more there are instances of uncontrollable bleeding in the young male. The duration of the ulcer is of much concern because the induration and vascularity surrounding the chronic lesion may be such to prevent immediate and complete cessation of the hemorrhage. Therefore, the duration is of importance as well as the age of the patient. It must be remembered that frequently exsanguination may occur from an acute ulceration which has given no previous symptoms.

Some confusion exists regarding indications for operation but there are three definite reasons, namely: (1) those patients who in spite of early and full blood replacement continue to bleed and blood volume cannot be maintained and the

pressure is never sustained, (2) those patients with massive bleeding and who after treatment cease to bleed and then massively hemorrhage before leaving the hospital, (3) those patients who continue to slowly bleed in spite of conservative treatment. This is particularly true of individuals past the age of 45 years and those with longstanding ulcer.

There are others requiring early operation but cannot be classified with a set formula. This would be impossible since each particular case is different and must be handled accordingly. Control of bleeding is mandatory before irreversible damage to vital organs has occurred. The continued giving of large quantities of blood rather than offering surgery to these patients, is not only to be condemned but is fraught with dangers of the complication of abnormal clotting seen following the transfusions.

At operation the objective is to quickly but effectively control the bleeding. This is not always easy since the cause of the bleeding cannot be found even after exploration. In the absence of a demonstrable ulcer an exploratory gastroduodenotomy is indicated. This may demonstrate lesions on the posterior wall or early small lesions which may have have been overlooked. Removal of the ulcer should be done when practicable but there are a few patients in whom this cannot be done. Suture plication can be successful and effectively followed by a definitive ulcer operation, preferably subtotal resection. Vagotomy with posterior gastroenterostomy or hemigastrectomy may be sufficient in the poor-risk patients.

When the cause for the bleeding has not been established, blind subtotal gastric resection has been considered indicated. There is inadequate proof to conclusively establish the merit of such a course, although many such cases have been reported.

J. D. MARTIN, JR., M.D.
Emory University Hospital
Emory University, Ga.

BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Operative Technic in Specialty Surgery. Edited by WARREN H. COLE, M.D. Appleton-Century-Crofts, Inc., New York, 1956. Second Edition $20.00

In 967 pages this volume represents a revision of the first edition bringing it up to date by some 32 authors. It is a companion volume for the one on operative technic in general surgery.

It covers the various surgical specialties in which a general surgeon might sometimes have to venture and the discussions of diagnosis and preoperative and postoperative treatment are very good. The illustrations as a whole are very good but those in the department of gynecology leave much to be desired.

This book is recommended for the general surgeon in the small communities where specialists are not available and/or for the general surgeon in large cities who might have to carry out some of these procedures in connection with some others.

W. P. NICOLSON, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Doctor and Patient and the Law. 3rd. ed. By Doctor Reagan, Member State Bar of California; Professor of Legal Medicine, College of Medical Evangelists; Clinical Professor of Forensic Medicine, School of Medicine, University of Southern California; Consulting Staff, Hollywood Presbyterian Hospital, Los Angeles, Methodist Hospital of Southern California, Los Angeles, Physicians and Surgeons Hospital, Glendale, California; and Member of the Staff, Los Angeles County Hospital, Los Angeles. St. Louis, The C. V. Mosby Company, 1956. $12.50.

Fractures of the Facial Skeleton. By N. L. ROWE, F.D.S.R.C.S. (Eng.), L.R.C.P. (Lond.), M.R.C.S. (Eng.), L.M.S.S.A. (Lond.), H.D.D.R.C.S. (Edin.), Consultant in Oral Surgery, Plastic and Maxillo-facial Surgery Unit, Rooksdown House, Basingstoke, Hants. Civilian Consultant Dental Surgeon to the Royal Navy, Honorary Civilian Lecturer in Maxillo-Facial Injuries to the Depot and Training Establishment, Royal Army Dental Corps, Aldershot, Hants; AND H. C. KILLEY, F.D.S.R.C.S. (Eng.) L.R.C.P. (Lond.), M.R.C.S. (Eng.), L.M.S.S.A. (Lond.), H.D.D.R.C.S. (Edin.), Consultant in Oral Surgery, Plastic and Maxillo-facial Surgery Unit, Rooksdown House, Basingstoke, Hants, Honorary Civilian Lecturer in Maxillo-Facial Injuries to the Depot and Training Establishment, Royal Army Dental Corps, Aldershot, Hants; with a Foreword by SIR REGINALD WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S.S. (Hon.), F.A.C.S. (Hon.) with 1231 illustrations. Baltimore, The Williams & Wilkins Company, 1955. $22.00.

The Diagnosis and Management of Urological Cases, A Handbook for Students, Residents and General Practitioners. By BRUCE W. T. PENDER, M.B., B.S., F.R.C.S., Senior Surgical Registrar, St. George's Hospital, London; Formerly Surgical Registrar, St. Thomas's Hospital, London; AND JAMES O. ROBINSON, M.A. (Cantab.) M.Chir., F.R.C.S., Senior

Surgical Registrar, St. Bartholomew's Hospital, London; Formerly Clinical Assistant, St. Peter's Hospital for Stone, London; foreword by SIR W. HENEAGE OGILVIE. K.B.E., M.A., M.Ch., F.R.C.S., Consulting Surgeon, Guy's Hospital, London, Late Maj.-Gen., A.M.S. Baltimore, The Williams & Wilkins Company, 1955. $5.00.

Body Fluids in Surgery. By A. W. WILKINSON, Ch.M., F.R.C.S.E., Senior Lecturer in Surgery, University of Aberdeen; Assistant Surgeon, Aberdeen Royal Infirmary and Royal Aberdeen Hospital for Sick Children, Formerly Lecturer in Surgery, University of Edinburgh, and Assistant Surgeon, Deaconess Hospital, Edinburgh. Baltimore, The Williams & Wilkins Company, 1955. $4.00.

Diagnosis and Treatment of Vascular Disorders (Angiology). Edited by SAUL S. SAMUELS, A.M., M.D., F.A.C.A., F.A.C.C., Editor-in-chief, Angiology; Pres., Angiology Research Foundation; Director of Angiology and Attending Vascular Surgeon, Brooklyn Hebrew Home and Hospital for the Aged; Chief, Dept. of Peripheral Arterial Diseases, Stuyvesant Polyclinic Hospital, N. Y.; Fellow in Surgery, N. Y. Academy of Medicine; Formerly Chief of Vascular Clinic and Adjunct Att. Surgeon, Bellevue Hospital, N. Y.: Honorary Member, Cuban Soc. of Angiology; Consulting Vascular Surgeon, Long Beach Memorial Hospital, Long Beach, N. Y.; Pres., Am. College of Angiology. Baltimore, The Williams and Wilkins Company, 1956. $16.00.

Skin Surgery. By ERVIN EPSTEIN, M.D., Assistant Clinical Professor of Medicine (Dermatology), Stanford University Medical School, Chief of Dermatology and Syphilology at Highland-Alameda County Hospital, Consultant to Oakland Area Veterans Hospital, Mt. Zion Hospital, Camp Parks AFB Hospital, Editorial Staff of "Dermatologica", Member of American Dermatological Association, American Academy of "Dermatology and Syphilology, Society for Investigative Dermatology, Abstract Staff of "Excerpta Medica", Diplomate of American Board of Dermatology and Syphilology, etc. Philadelphia, Lea & Febiger, 1956. $7.50.

The Cervical Syndrome. By RUTH JACKSON, B.A., M.D., F.A.C.S., Clinical Assistant Professor of Orthopaedic Surgery, Southwestern Medical School of the University of Texas, Dallas; Attending Orthopaedic Surgeon, Baylor University Hospital, Formerly Chief of Orthopaedic Surgery, Parkland Hospital and Instructor in Orthopaedic Surgery, Baylor University College of Medicine, Dallas, Texas. Springfield, Illinois, Charles C Thomas. $4.75.